# A PROSPECTIVE ECHOCARDIOGRAPHIC STUDY IN PATIENTS WITH HYPOTHYROIDISM AND HYPERTHYROIDISM

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# CERTIFICATE

This is to certify that this dissertation titled "**A PROSPECTIVE ECHOCARDIOGRAPHIC STUDY IN PATIENTS WITH HYPOTHYROIDISM AND HYPERTHYROIDISM"** submitted by **DR.S.SIVARAMASUBRAMANIAN** to the faculty of General Medicine, The Tamilnadu Dr. M.G.R. Medical University, Chennai in partial fulfillment of the requirement for the award of MD degree (branch I) General Medicine, is a bonafide research work carried out by him under our direct supervision and guidance.


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# DECLARATION

I**, DR.S.SIVARAMASUBRAMANIAN,** solemnly declare that the dissertation titled "**A PROSPECTIVE ECHOCARDIOGRAPHIC STUDY IN PATIENTS WITH HYPOTHYROIDISM AND HYPERTHYROIDISM"** has been prepared by me. This is submitted to **The Tamilnadu Dr. M.G.R. Medical University, Chennai,** in partial fulfillment of the regulations for the award of MD degree (branch I) General Medicine.

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# CONTENTS

# 1.INTRODUCTION

The thyroid gland and the heart share a close relationship arising in embryology. In ontogeny the thyroid and heart anlage migrate together. The close physiological relationship is affirmed by predictable changes in cardiovascular function across the entire range of thyroid disease states.

In fact, cardiovascular manifestations are some of the most common and characteristic findings of hyperthyroidism. In a tropical country like India where the iodine deficiency status is proficient, hypothyroidism silently exerts its morbid effects in a large group of populations. As per an extrapolated statistics published by American Medical Women Association there are **78,314,013** cases, out of **1,065,070,607** studied populations in India.

Where Pediatricians worry about the mental status of children and Obstetricians worry about the menstrual irregularities and infertility caused by hypo and hyperthyroidism, Physicians and Cardiologists started worrying about the adverse cardiovascular effects of thyroid deficiency and excess.

According to one study from AIIMS (All India Institute of Medical Sciences), in India about 40 million people are suffering from thyroid

disorders. In one hand the urbanization and changes in lifestyle increase the cardiovascular morbidity and mortality in younger populations, the current threat is sub clinical hypothyroidism and its adverse effects on cardiovascular system.

Not only by its atherogenic effect over the blood vessels and lipid profile, but also through so many different effects on the heart and hemodynamics, hypothyroidism has increased the cardiovascular morbidity and mortality.

Hyperthyroidism is an arrythmogenic state and it also exerts various functional and anatomical effects in the CVS.

Lack of large population study regarding the cardiovascular morbidity in thyroid disorders and lack of awareness of hyper and hypothyroidism among undereducated people give way for thyroid disorders to manipulate various systems silently long before they come to light.

So, hypothyroidism or hyperthyroidism can primarily cause so many systemic manifestations or they act as worst co morbid conditions with other CVS disorders.

One interesting point to bear in mind is not only the thyroid disorders affect the heart but the drugs used to treat arrhythmias like Amiodarone can also adversely affect the thyroid status.

Some special situations like pregnancy, CCF and CRF are all put forth an extra burden over thyroid and a vicious cycle may set in.

Early observations of the heart in hyperthyroidism emphasize the similarity to that of hyperadrenergic states and moreover propose enhanced sensitivity to catecholamines in this setting. This postulate forms the basis for the test described by **Goetsch in 1918** in which hyperthyroidism could be diagnosed by demonstrating a marked cardio acceleration and blood pressure response to small, subcutaneous doses of epinephrine. Hyperthyroid subjects have decreased circulating catecholamine concentrations despite the appearance of increased adrenergic signs and symptoms.

The role of physician in this multidisciplinary approach is to mainly concentrate on the cardiovascular morbidity while treating or correcting the thyroid aberrancy.

Though being protected from major coronary events prior to menopause, where females outnumber males in having thyroid disorders, which toggles the protective effect of estrogen. This is how one hormone influences the protective effect of the other.

The molecular biology astonishingly throws light over many of the hidden mechanisms, the primary and secondary role of thyroid hormone and the resultant pathophysiology in excess and deficient status of the same

hormone are explored in full. Moreover early detection of thyroid dysfunction accurately by RIA (Radio Immuno Assay) and other techniques made the iceberg to come to the surface.

N.Kochupillai, an eminent Endocrinologist from All India Institute of Medical Sciences (AIIMS), states that tropical countries should try to eliminate the iodine deficiency by redirecting the Health Resources, so as to reduce the resultant hypothyroidism and its hemodynamic burden.

# 2. REVIEW OF LITERATURE

To identify and manage thyroid hormone mediated cardiac disease states, it is important to understand the cellular mechanisms of thyroid hormone on the heart and vascular smooth muscle cells [2]

## 2.1.0 CELLULAR MECHANISMS OF THYROID HORMONE ACTION

### 2.1.1 Regulation of thyroid hormone synthesis and action

Under the regulation of thyroid stimulating hormone (thyrotropin, TSH), the thyroid gland has the unique property of concentrating serum iodide and through a series of enzymatic steps synthesizes predominantly tetraiodothyronine ($T_4$ 85 percent) and a smaller percentage of triiodothyronine ($T_3$ 15 percent).[3] The major source of $T_3$ synthesis is by conversion by 5′ monodeiodination primarily in the liver and to a lesser degree in the kidney.[4] A variety of studies have confirmed $T_3$ as the active form of thyroid hormone that accounts for the vast majority of biological effects including stimulation of tissue thermogenesis, alterations in the

expression of various cellular protein, and actions on the heart and vascular smooth muscle cells. [2] [6]

Serum-free $T_3$ in turn is taken up by a process of facilitated diffusion within cells, where it appears to pass without additional protein binding to the cell nucleus. Most data indicate that the cardiac myocyte cannot metabolize $T_4$ to $T_3$. Therefore despite the presence of the relevant enzymes, all of the observed nuclear actions and changes in gene expression result from changes in blood levels of $T_3$.

### 2.1.2Thyroid hormone Receptors (TRs)in the Myocytes

The cardiac myocyte expresses both the alpha and beta isoforms of the thyroid hormone receptors (TRs), which arise from two separate genes. These genes give rise to splice variants $TRalpha_1$ and $TRalpha_2$, of which only the former binds thyroid hormone, as well as $TRbeta_1$, $TRbeta_2$, and $TRbeta_3$.[5]

As reported for the steroid and retinoic acid family of receptor proteins, the TRs act by binding as either homodimers or heterodimers to the thyroid hormone response elements (TREs) in a promoter region of specific

genes.[5] Binding to the promoter regions can either activate or repress gene expression. [6]

### 2.1.3 Molecular mechanism of thyroid hormone action on heart

Thyroid hormone transcriptionally regulates many cardiac proteins. They include structural and regulatory proteins, as well as a variety of cardiac membrane ion channels and cell surface receptors, thus providing a molecular mechanism to explain many of the diverse effects of thyroid hormone on the heart. The first reported and the best studied to date has been the myosin heavy chain isoforms alpha and beta). The human ventricle expresses primarily beta myosin, and there appear to be few, if any, alterations in isoform expression accompanying thyroid disease states.

Changes in myosin heavy chain isoform expression occur in the human atria in a variety of disease states including congestive heart failure, and whether these changes are thyroid hormone mediated remains to be determined.[7]

The sarcoplasmic reticulum calcium-activated ATPase is an important ion pump that determines the magnitude of myocyte calcium cycling. The reuptake of calcium into the sarcoplasmic reticulum early in diastole in part

determines the rate at which the left ventricle relaxes (isovolumetric relaxation time, IVRT).[1] The activity of SERCA2 in turn is regulated by the polymeric protein phospholamban with its ability to inhibit SERCA activity further modified by the level of phosphorylation of the individual phospholamban monomers.[8] Inotropic agents that enhance cardiac contractility through increases in myocyte cAMP do so by stimulating the phosphorylation of phospholamban. Thyroid hormone inhibits the expression of phospholamban and increases phospholamban phosphorylation.[9]

Thyroid hormone exerts most of its direct effects on cardiac contractility by regulating calcium cycling through the SERCA-phospholamban system both transcriptionally and posttranscriptionally. This molecular mechanism can explain why diastolic function varies inversely across the entire spectrum of thyroid disease states including even mild, subclinical hypothyroidism. [2] [11]

Changes in other myocyte genes including $Na^+/K^+$ ATPase account for the increase in basal oxygen consumption of the experimental hyperthyroid heart and explain the decrease in digitalis sensitivity of hyperthyroid patients. A variety of studies have shown that thyroid hormone

can regulate the genetic expression of its own nuclear receptors within the cardiac myocyte.

### 2.1.4 Nongenomic mechanisms of thyroid hormone action

In addition to the well-characterized nuclear effects of thyroid hormone, a growing body of cardiac responses to thyroid hormone appear to be mediated through nongenomic mechanisms[12] as suggested by their relatively rapid onset of action (faster than can be accounted for by changes in gene expression and protein synthesis) and failure to be affected by inhibitors of gene transcription. The significance of these diverse actions remains to be established but may explain the ability of acute $T_3$ treatment to alter cardiovascular hemodynamics. They may alter the functional properties of membrane ion channels and pumps including the sodium channel and the inward rectifying potassium current ($I_k$).

## 2.2.0 THYROID FUNCTION TESTING

A number of sensitive and specific laboratory tests can establish a diagnosis of thyroid disease with a high degree of precision. Serum TSH is the most widely used and most sensitive measure for the diagnosis of both hypothyroidism and hyperthyroidism. [13] Serum TSH levels uniformly

increase (>5 mIU/ml) in patients with primary hypothyroidism, and conversely, because of the normal feedback of excess levels of $T_4$ (and $T_3$) on the pituitary synthesis and secretion of TSH, the levels are low (<0.04 to 0.01 mIU/ml) in hyperthyroidism. Measures of free $T_4$ can be useful when coexistent hepatic, nutritional, or genetic disease may alter thyroxine-binding globulin content. Autoimmune thyroid disease (Hashimoto and Graves) can be further diagnosed by the use of serologic measures of antithyroid antibodies, most specifically antithyroid peroxidase (anti-TPO) or antithyroglobulin antibodies.

## 2.3.0 THYROID HORMONE–CATECHOLAMINE INTERACTION

Early observations of the heart in hyperthyroidism emphasize the similarity to that of hyperadrenergic states and moreover propose enhanced sensitivity to catecholamines in this setting. Increased $beta_1$ adrenergic receptors on cardiac myocytes observed in experimental hyperthyroidism provide a mechanism for enhanced catecholamine sensitivity. A recent carefully controlled study of subhuman primates, however, found no increase in sensitivity of the heart or cardiovascular system to catecholamines in experimental hyperthyroidism.[14]

Accompanying the increased levels of $beta_1$-adrenergic receptors and guanosine triphosphate binding proteins, thyroid hormone decreases the expression of cardiac-specific (V, VI) adenylyl cyclase catalytic subunit isoforms and thereby maintains cellular response to beta-adrenergic agonists within normal limits.

Table 2.1-- **Thyroid Hormone Regulation of Cardiac Gene Expression**

| **Positively Regulated** | **Negatively Regulated** |
|---|---|
| Alpha-myosin heavy chain | Beta-myosin heavy chain |
| Sarcoplasmic reticulum $Ca^{2+}$ -ATPase | Phospholamban |
| $Na^+$, $K^+$ -ATPase | $Na^+/Ca^{2+}$ exchanger |
| Voltage-gated potassium channels (Kv1.5, Kv4.2, Kv4.3) | Thyroid hormone receptor alpha1 |
| Atrial and brain natriuretic peptide | Adenylyl cyclase (AC) types V, VI |
| Malic enzyme | Guanine nucleotide–binding protein $G_i$ |
| Beta-adrenergic receptor | |

## 2.4.0 Hemodynamic Alterations in Thyroid Disease

Changes in myocardial contractility and cardiovascular hemodynamics occur across the entire spectrum of thyroid disease.[11]

## 2.4.1 Effects of T3 on heart and vascular smooth muscle

Multiple studies including those in experimental animals, as well as invasive and noninvasive measurements in patients, indicate that triiodothyronine regulates cardiac inotropy and chronotropy through a variety of both direct and indirect mechanisms. [12] [17] Direct effects on vascular smooth muscle cells decrease systemic vascular resistance of the arterioles of the peripheral circulation. [17] [20] A decrease in mean arterial pressure and activation of the renin-angiotensin-aldosterone system occurs, as does an increase in renal sodium reabsorption. The increase in plasma volume coupled with an increase in erythropoietin leads to an increase in blood volume and a rise in cardiac preload.[18]

Thus a combination of lower systemic vascular resistance (by as much as 50 percent), coupled with increases in venous return and preload, increases cardiac output.

TABLE2. 2- **Cardiovascular Changes with Thyroid Disease**

| **Parameter** | **Normal** | **Hyperthyroid** | **Hypothyroid** |
|---|---|---|---|
| Systemic vascular resistance (dyne-cm · $sec^{-5}$) | 1500-1700 | 700-1200 | 2100-2700 |
| Heart rate (beats/min) | 72-84 | 88-130 | 60-80 |
| Cardiac output (liter/min) | 5.8 | >7.0 | <4.5 |
| Blood volume (% of normal) | 100 | 105.5 | 84.5 |

Triiodothyronine appears to reduce systemic vascular resistance by both direct effects on vascular smooth muscle cells and through changes in the vascular endothelium potentially involving the synthesis and secretion of nitric oxide.[16] Thus the combination of increased cardiac output and decreased arterial compliance, which may be more pronounced in older patients with some degree of arterial vascular disease, leads to systolic hypertension in up to 30 percent of patients.[17]

### 2.4.2 Hemodynamic alterations in hypothyroidism

In hypothyroidism, systemic vascular resistance may increase as much as 30 percent. Mean arterial pressure rises with up to 20 percent of patients having significant diastolic hypertension.[17] Even mild hypothyroidism may decrease endothelial-derived relaxing factors.[21] The diastolic hypertension of hypothyroidism is frequently associated with a low renin level and a decrease in hepatic synthesis of renin substrate. This leads to a characteristically low level of salt sensitivity, again reinforcing the importance of an increase in systemic vascular resistance underlying the mechanism for diastolic hypertension.[22]

## 2. 5.0Hemodynamic alterations in Hyperthyroidism

### 2.5.1 Exercise Intolerance

Cardiovascular symptoms are an integral and often the predominant clinical presentation of patients with hyperthyroidism. Palpitations resulting from both an increase in the rate and force of cardiac contractility are present in the majority of patients. The increase in heart rate results from both an increase in sympathetic tone and a decrease in parasympathetic stimulation. Heart rates >90 beats per minute both at rest and during sleep commonly

occur, the normal diurnal variation in heart rate is blunted and the increase during exercise is exaggerated.

Many hyperthyroid patients experience exercise intolerance and exertional dyspnea, due in part to weakness in skeletal and respiratory muscle. [2] [24] In the setting of a low vascular resistance and increased preload, cardiac functional reserve is compromised and cannot further rise to accommodate the demands of submaximal or maximal exercise.[23]

### 2.5.2-Chestpain in thyrotoxicosis

Patients can experience angina-like chest pain. In older patients with known or suspected coronary artery disease, the increase in cardiac work associated with the increase in cardiac output and cardiac contractility of hyperthyroidism can produce myocardial ischemia, which can respond to beta-adrenergic blocking agents or the restoration of a euthyroid state.

In rare patients, usually younger women, there is a syndrome of chest pain at rest associated with ischemic ECG changes. Cardiac catheterization has demonstrated that the majority of these patients have angiographically normal coronary arteries; however, coronary vasospasm has been reported similar to that found in variant angina. Myocardial infarction rarely

develops, and these patients appear to respond to calcium channel blockers or to nitroglycerin.

### 2.5.3 PHT in Thyrotoxicosis

Recent reports have shown that hyperthyroidism is associated with a significant degree of pulmonary hypertension (pulmonary artery systolic pressure >75 mm Hg), which was reversible after treatment of the Graves disease. This observation implies that although systemic vascular resistance is decreased with thyrotoxicosis, peripheral vascular resistance is not. Perhaps all patients with unexplained pulmonary hypertension should be evaluated for thyroid disease with measurement of serum TSH. [18] [25]

### 2.6.0 Atrial fibrillation and Hyperthyroidism

The most common rhythm disturbance in patients with hyperthyroidism is sinus tachycardia.[3] Its clinical impact, however, is overshadowed by patients with atrial fibrillation resulting from thyrotoxicosis. The prevalence of atrial fibrillation and the less common forms of supraventricular tachycardia in this disease ranges from 2 to 20 percent. [26] [27] When compared with a control population with normal thyroid function and a prevalence of atrial fibrillation of 2.3 percent, the

prevalence of atrial fibrillation in overt hyperthyroidism was 13.8 percent.[26] In a study of more than 13,000 hyperthyroid patients, the prevalence rate for atrial fibrillation was less than 2 percent, perhaps because of earlier recognition and disease treatment. When that same group of patients was analyzed for age distribution, it was seen that there was a stepwise increase in prevalence in each decade peaking at approximately 15 percent in patients older than 70 years.[25] This latter study confirms essentially all reports that atrial fibrillation caused by hyperthyroidism is more common with advancing age. Thus the yield of abnormal thyroid function testing including a low serum TSH appears to be low in patients with new-onset atrial fibrillation. However, the ability to restore thyrotoxic patients to a euthyroid state and sinus rhythm justifies TSH testing in most patients with the recent onset of otherwise unexplained atrial fibrillation.

Treatment of atrial fibrillation in the setting of hyperthyroidism includes beta-adrenergic blockade using one of a variety of $beta_1$ selective or nonselective agents to control the ventricular response. This symptomatic measure can be accomplished rapidly, whereas the treatments leading to restoration of the euthyroid state require more time. Anticoagulation in patients with hyperthyroidism and atrial fibrillation is controversial. [22] [28]

The potential for systemic or cerebral embolization must be weighed against the risk of bleeding and complications related to this therapy.

Successful treatment of hyperthyroidism with either radioiodine or antithyroid drugs and restoration of normal serum levels of $T_4$ and $T_3$ are associated with reversion to sinus rhythm in two thirds of patients within 2 to 3 months. [25]

In older patients or in the setting of atrial fibrillation of longer duration, the rate of reversion to sinus rhythm is lower and therefore electrical or pharmacological cardioversion should be attempted, but only after the patient has been rendered euthyroid.

## 2.7.0 HEART FAILURE AND HYPERTHYROIDISM

The cardiovascular alterations in hyperthyroidism include increased resting cardiac output and enhanced cardiac contractility. Nevertheless, a minority of patients present with symptoms including dyspnea on exertion, orthopnea, and paroxysmal nocturnal dyspnea, as well as signs demonstrating peripheral edema, neck vein distention, and an $S_3$ indicative of heart failure. This complex of findings coupled with a failure to increase

the LV ejection fraction with exercise has suggested the possibility of a hyperthyroid cardiomyopathy.

The term often used in this setting—high output failure—is not appropriate because although resting cardiac output is as much as two to three times normal, the exercise intolerance does not appear to be a result of cardiac failure but rather of skeletal muscle weakness. [2] [24] High output states, however, can increase renal sodium reabsorption, expand plasma volume, and cause development of peripheral edema, pleural effusions, and venous hypertension. Interestingly, whereas systemic vascular resistance falls with hyperthyroidism, the pulmonary vascular bed is not similarly affected and, as a result of the increase in output to the pulmonary circulation, there is an increase in pulmonary artery pressures. [18] [25] This results in a rise in mean venous pressure, neck vein distention, hepatic congestion, and peripheral edema of the type associated with primary pulmonary hypertension or right heart failure. [2] [18] [25]

Patients with longstanding hyperthyroidism and marked sinus tachycardia or atrial fibrillation can develop low cardiac output, impaired cardiac contractility with a low ejection fraction, an $S_3$ and pulmonary congestion, all consistent with congestive heart failure.[1] Review of such

cases suggests that impairment in left ventricular function results from prolonged high heart rate and the development of rate-related heart failure. When the left ventricle becomes dilated, mitral regurgitation may also develop. Recognition of this entity is important because treatments aimed at slowing heart rate or controlling the ventricular response in atrial fibrillation appear to improve left ventricular function even before initiation of antithyroid therapy.[1]

Because these patients are critically ill they should be managed in an intensive care unit setting. Some patients with hyperthyroidism (similar to the overall congestive heart failure population) do not tolerate initiation of beta-adrenergic blocking drugs in full doses, and treatment can be started with lower doses of short-acting beta-blocking drugs in conjunction with classic forms of treatment of acute congestive heart failure including diuresis.

The increase in rate-pressure product and oxygen consumption that results from hyperthyroidism can impair cardiac function in older patients with known or suspected ischemic, hypertensive, or valvular heart disease.

## TREATMENT

Treatment of patients with thyrotoxic cardiac disease should include a beta-adrenergic antagonist to lower the heart rate to 10 or 15 percent above normal. This will cause the tachycardia-mediated component of ventricular dysfunction to improve, whereas the direct inotropic effects of thyroid hormone will persist.[1] The rapid onset of action and the improvement in many of the signs and symptoms of hyperthyroidism indicate that most patients with overt symptoms should receive beta-blocking agents. Definitive therapy can then be accomplished safely with iodine-131 alone or in combination with an antithyroid drug.

## 2.8.0 HYPOTHYROIDISM

In contrast to the dramatic clinical signs and symptoms of hyperthyroidism, the cardiovascular findings of hypothyroidism are more subtle.[31] Mild degrees of bradycardia, diastolic hypertension, a narrow pulse pressure and a relatively quiet precordium, and decreased intensity of the apical impulse are characteristic.

### 2.8.1 Hemodynamic changes in hypothyroidism

Hemodynamic changes of hypothyroidism are diametrically opposite to that of hyperthyroidism and explain many of the physical findings.

Despite the decrease in cardiac output and contractility of the hypothyroid myocardium, recent studies of myocardial metabolism by PET scan have shown energy inefficiency of the hypothyroid myocardium.

The oxygen cost of work increases primarily as a result of the increase in afterload.[32] Treatment of hypothyroid patients with the restoration of a euthyroid state resolves these changes in parallel with a return of systemic vascular resistance to lower levels. [32] [33]

### 2.8.2 Changes in Lipid profile

Hypothyroidism also produces increases in total and low-density lipoprotein (LDL) cholesterol in proportion to the rise in serum TSH.[33] Although thyroid hormone can alter cholesterol metabolism through multiple mechanisms including a decrease in biliary excretion, it appears that changes in LDL metabolism caused by decreases in LDL receptor number are a primary mechanism. [35] [36]

### 2.8.3 Pericardial effusion in Hypothyroidism

Pericardial effusions can occur consistent with observation that patients with hypothyroidism have an increase in volume of distribution of albumin and a decrease in lymphatic clearance function. Occasionally the

pericardial effusions are quite large, causing the appearance of cardiomegaly on chest radiograph. Although rare, tamponade with hemodynamic compromise can occur. Echocardiography demonstrates small to moderate effusions in up to 30 percent of overtly hypothyroid patients, which resolve over a period of weeks to months after initiation of thyroid hormone replacement.[31]

### 2.8.4 ECG in Hypothyroidism

As a result of changes in ion channel expression, the ECG in hypothyroidism is characterized by **sinus bradycardia**, **low voltage**, and **prolongation of the action potential duration and the QT interval**. The latter in turn predisposes the patients to ventricular arrhythmias, and cases of patients with acquired **torsades de pointes** that have improved or completely resolved with hormone replacement have been reported. (31)

### 2.8.5 CAD risk in Hypothyroidism

As a result of increases in risk factors including hypercholesterolemia, hypertension, and elevated levels of homocysteine, patients with hypothyroidism may have increased risk for atherosclerosis and coronary and systemic vascular disease. [34] [35] Recent studies have shown increases in

abdominal aortic atherosclerosis in elderly women patients with even mild hypothyroidism.[36]

Whether patients with hypothyroidism have an increase in coronary artery disease is an important clinical issue. Recent findings suggest increased cardiovascular morbidity and mortality with untreated subclinical hypothyroidism.[37] Noninvasive studies including thallium scanning have demonstrated abnormalities in perfusion suggestive of myocardial ischemia, but these defects appear to resolve with thyroid hormone treatment.

In patients younger than 50 years of age with no history of heart disease, it is possible to initiate full replacement doses of l-thyroxine (100 mg to 150 mg per day) without concern for untoward cardiac effects. In patients older than age 50 with known or suspected coronary artery disease, the issue is more complicated.

**2.9.0 DIAGNOSIS**

Hashimoto disease, postradioiodine therapy for Graves disease, and iodine deficiency (in parts of the world where that remains a public health problem) are the leading causes of hypothyroidism and produce diagnostic elevation in serum TSH.[13] Thus the finding of an elevated TSH is sufficient

to establish the diagnosis and form the basis for treatment. In routine practice additional testing with a serum $T_4$ and $T_3$ resin uptake is confirmatory.

The prevalence of hypothyroidism is estimated at 3 to 4 percent for overt disease and 7 to 10 percent for the milder forms of disease. Thus TSH screening can be advised for all adults and particularly in patients with hypertension, hypercholesterolemia, hypertriglyceridemia, coronary or peripheral vascular disease, and unexplained pericardial or pleural effusions and for a variety of musculoskeletal syndromes or statin-associated myopathy. [4] [40]

## 2.10.0 TREATMENT

The response to treatment of hypothyroidism is predictable, especially from a cardiovascular perspective. Stepwise thyroid hormone replacement using levothyroxine sodium (Levoxyl, Synthroid) incrementally decreases serum TSH, serum cholesterol, and serum creatine kinase and improves left ventricular performance. Full replacement is accomplished when serum TSH is normal.[13] In the rare condition of myxedema coma, which is characterized by patients with severe and longstanding hypothyroidism who develop hypothermia, altered mental status, hypotension, bradycardia, and

hypoventilation, the need for thyroid hormone replacement is more emergent and treatment can be accomplished with either 100 mg a day of T4 or 25 to 50 mg a day of $T_3$ administered intravenously. These patients often require intensive care unit monitoring with volume repletion, gentle warming, and ventilatory support in the face of $CO_2$ retention. Administration of hydrocortisone (100 mg q8h) should be undertaken until results of serum cortisol testing are obtained. When treated in this manner, hemodynamics including systemic vascular resistance, cardiac output, and heart rate improve within 24 to 48 hours.

## 2.11.0 SUBCLINICAL THYROID DISEASE

In contrast to overt symptomatic thyroid disease, subclinical thyroid disease implies the absence of classic hyperthyroid or hypothyroid related symptoms in patients with thyroid dysfunction. The definition has been further refined to include the demonstration of an abnormal TSH level in the face of normal serum levels of total $T_4$, free $T_4$, total $T_3$ and free $T_3$. [14] [41] With the advent of widespread TSH screening, the magnitude of subclinical thyroid disease may exceed that of overt disease by threefold to fourfold.[35]

### 2.11.1 SUBCLINICAL HYPOTHYROIDISM

Subclinical hypothyroidism defined as a TSH above the upper range of the reference population (usually >5 mIU/ml) is seen in up to 9 percent of unselected populations, and clearly prevalence increases with advancing age.[35] In contrast to younger patients in whom there is a strong female predilection, in older populations, this difference is lost.

Subclinical hypothyroidism alters lipid metabolism, atherosclerosis, cardiac contractility, and systemic vascular resistance. Cholesterol levels rise in parallel with increments in TSH elevations starting at 5 mIU/liter. A large study of women in Rotterdam showed that atherosclerosis and myocardial infarction increased with odds ratios of 1.7 and 2.3 in subclinical hypothyroid women, respectively. Interestingly, the presence of antithyroid antibodies as a measure of autoimmune thyroid disease indicated heightened risk.[36] Restoration of serum TSH to normal after thyroid hormone replacement improved lipid levels, lowered systemic vascular resistance, and improved cardiac contractility.[41] Patients with subclinical hypothyroidism have prolonged isovolumic relaxation times, whereas systolic contractile function does not change. Replacement with l-thyroxine sodium at a mean dose of 68 mg per day (range 50 to 100 mg per day) restored isovolumic

relaxation times to normal and when compared with the same patients before therapy, systemic vascular resistance declined and systolic function significantly improved.[42]

A variety of studies have indicated that the changes in systemic vascular resistance result from alterations in endothelium-dependent vasodilation. [11] [22] Taken together, it seems appropriate to recommend thyroid hormone replacement for all patients with subclinical hypothyroidism from a cardiovascular perspective. The lack of untoward cardiac effects observed when serum TSH levels normalize indicate that the potential benefits far outweigh the risks of treatment. [2] [11] [34]

### 2.11.2 SUBCLINICAL HYPERTHYROIDISM

Subclinical hyperthyroidism is diagnosed when serum TSH is low (<0.1 mIU/ml) and both $T_4$ and $T_3$ are normal.[13] The significance of subclinical hyperthyroidism was conclusively established from a study of atrial fibrillation in patients 60 years of age or older in the Framingham cohort.[43] Prevalence of atrial fibrillation after 10 years was 28 percent in the subclinical hyperthyroid patient population compared with 11 percent in patients with normal thyroid function with a relative risk of 3.1.

A large U.S. study of patients 65 years or older confirmed and extended this result.[44] A population-based study of more than 1000 individuals with subclinical hyperthyroidism not receiving l-thyroxine therapy or antithyroid medication demonstrated that a TSH level of less than 0.5 was associated with twofold increased mortality with relative risk of 2.3 to 3.3 from all causes, which in turn was largely accounted for by increases in cardiovascular mortality.[44]

# 3. AIMS AND OBJECTIVES

1. To study the effects of Hypothyroidism and Hyperthyroidism in CVS using 2D Echo (TTE)

2. To correlate the various Echo dimensions in these disorders with age, sex and T4 levels.

3. To compare the outcome with the international studies.

# 4. MATERIALS AND METHODS

**Duration of study:** 6 months

**Period of study**: February 2009 to July 2009

**Selection of study subjects:** Patients attending Department of Medicine and Department of Endocrinology, Government Rajaji Hospital, Madurai with Hypothyroidism or Hyperthyroidism

**Number of Patients studied:** 50 patients in Hypothyroidism group and 50 patients in Hyperthyroidism group

**Data Collection:** Clinical, Biochemical and Echocardiographic Data.

**Methods:** Standard clinical and Laboratory methods.

**Ethical clearance:** Obtained

**Consent:** Informed consent obtained

**Inclusion Criteria:**

1. Overt or subclinical hypothyroidism

2. Overt or subclinical hyperthyroidism

3. Patients presented for the first time with Hypothyroidism or Hyperthyroidism

4. Patients without any other co morbid conditions

**Exclusion criteria:**

1. Hypertension

2. Diabetes

3. Coronary Artery Disease

4. Anemia

5. Patient already on treatment

6. Known valvular Heart Disease patients

7. COPD patients

8. Age >70years

9. Pregnancy

10. Patients taking drugs which could alter the cardiac functions such as beta blockers, calcium channel blockers and amiodarone

**Design of Study**: Prospective study

**Statistical Tools used:** Statistical analysis using **EPI-INFO 2002** desinged by CDC, Atlanta

**Echo done by:** 2D TTE – Aloka Full HD, using 3.5 MHz phased array transducer.

Measurements taken as per the recommendations of the American Society for Echocardiography.

# 5. RESULTS

Over 50 hypothyroid patients and 50 hyperthyroid patients had been selected for study. 2D Trans Thoracic Echo (TTE) done for them. Various parameters like LVEF, LV mass, IVS(s), IVS (d), presence of pericardial effusion, presence of DCM, MVP were recorded. The parameters were analyzed carefully using **EPI INFO 2002.**

**Table 1. Group statistics of Hypothyroidism and Hyperthyroism in our study**

| | Group | N | Mean | Std. Deviation | Std. Error Mean | T Test ( P Value) |
|---|---|---|---|---|---|---|
| Age | Hypothyroidism | 50 | 32.08 | 8.383 | 1.186 | 0.357 |
| | Hyperthyroidism | 50 | 30.74 | 5.872 | .830 | |
| LVEF% | Hypothyroidism | 50 | 58.46 | 11.67277 | 1.65078 | 0.007*** |
| | Hyperthyroidism | 50 | 63.12 | 2.70781 | .38294 | |
| IVS(s) | Hypothyroidism | 50 | .8820 | .19555 | .02766 | .906 |
| | Hyperthyroidism | 50 | .8780 | .13893 | .01965 | |
| IVS(d) | Hypothyroidism | 50 | .7280 | .20508 | .02900 | .621 |
| | Hyperthyroidism | 50 | .7100 | .15419 | .02181 | |
| | Hyperthyroidism | 50 | 3.1420 | .13262 | .01876 | |
| | Hyperthyroidism | 50 | 5.08 | .12778 | .01807 | |
| LVID(s) | Hypothyroidism | 50 | 3.72 | .30971 | .04380 | 0.120 |
| | Hyperthyroidism | 50 | 3.63 | .26128 | .03695 | |
| LVID(d) | Hypothyroidism | 50 | 5.088 | .56266 | .07957 | 0.173 |
| | Hyperthyroidism | 50 | 4.9420 | .49820 | .07046 | |
| LV mass inedex | Hypothyroidism | 50 | 124.44 | 45.39584 | 6.41994 | 0.111 |
| | Hyperthyroidism | 50 | 111.48 | 34.44857 | 4.87176 | |

The p value showed that the variables were comparable within the groups.

**Table2. Incidence of DCM in hypothyroidism and hyperthyroidism in our study**

| DCM | GROUP | | TOTAL | CHI SQUARE |
|---|---|---|---|---|
| | HYPOTHYROIDISM | HYPERTHYROIDISM | | |
| ABSENT | 41 | 50 | 91 | 0.003*** |
| DCM PRESENT | 9 | 0 | 9 | |
| TOTAL | 50 | 50 | 100 | |

9 out of 50 patients in Hypothyroidism group had DCM and none had DCM in hyperthyroidism group.

**Table3. Incidence of MVP in the study groups**

| MVP | | Group | | Total |
|---|---|---|---|---|
| | | Hypothyroidism | Hyperthyroidism | |
| Absent | Count | 49 | 43 | 92 |
| | % within Group | 98.0% | 86.0% | 92.0% |
| | % of Total | 49.0% | 43.0% | 92.0% |
| MVP present | Count | 1 | 7 | 8 |
| | % within Group | 2.0% | 14.0% | 8.0% |
| | % of Total | 1.0% | 7.0% | 8.0% |
| Total | Count | 50 | 50 | 100 |
| | % within Group | 100.0% | 100.0% | 100.0% |
| | % of Total | 50.0% | 50.0% | 100.0% |

Chi Square Test: p Value-0.29

7 out of 50 patients in Hypothyroidism group had MVP. The prevalence was 14% within the group. One patient out of 50 hypothyroid patients had MVP; hence the prevalence was 2% within the group.

**Table 4. Incidence PHT in the study groups**

| PHT | | Group | | Total |
|---|---|---|---|---|
| | | Hypothyroidism | Hyperthyroidism | |
| Absent | Count | 41 | 50 | 91 |
| | % within Group | 82.0% | 100.0% | 91.0% |
| | % of Total | 41.0% | 50.0% | 91.0% |
| PHT Present | Count | 9 | 0 | 9 |
| | % within Group | 18.0% | .0% | 9.0% |
| | % of Total | 9.0% | .0% | 9.0% |
| Total | Count | 50 | 50 | 100 |
| | % within Group | 100.0% | 100.0% | 100.0% |
| | % of Total | 50.0% | 50.0% | 100.0% |

Chi Square Test: p Value=0.003**

9 out of 50 patients in Hypothyroidism had PHT. This prevalence was 18% within the group. No one in hyperthyroidism group had PHT.

**Table 5. Incidence of pericardial effusion in the study group**

| Pericardial effusion | | Group | | Total |
|---|---|---|---|---|
| | | Hypothyroidism | Hyperthyroidism | |
| Absent | Count | 41 | 50 | 91 |
| | % within Group | 82.0% | 100.0% | 91.0% |
| | % of Total | 41.0% | 50.0% | 91.0% |
| Mild Effusion | Count | 2 | 0 | 2 |
| | % within Group | 4.0% | .0% | 2.0% |
| | % of Total | 2.0% | .0% | 2.0% |
| Moderate Effusion | Count | 7 | 0 | 7 |
| | % within Group | 14.0% | .0% | 7.0% |
| | % of Total | 7.0% | .0% | 7.0% |
| Total | Count | 50 | 50 | 100 |
| | % within Group | 100.0% | 100.0% | 100.0% |
| | % of Total | 50.0% | 50.0% | 100.0% |

Chi Square Test: p Value=0.007**

9 out of 50 patients with hypothyroidism had Pericardial Effusion. In those patients with pericardial effusion 14% had Moderate effusion within the group and 4% had mild effusion.

**Table 6. Age distribution within Hypothyroidism and Hyperthyroidism**

| AGE | | GROUP | | TOTAL |
|---|---|---|---|---|
| | | HYPOTHYROIDISM | HYPERTHYROIDISM | |
| < 20 YRS | COUNT | 1 | 1 | 2 |
| | % WITHIN GROUP | 2.0% | 2.0% | 2.0% |
| | % OF TOTAL | 1.0% | 1.0% | 2.0% |
| 20-30 YRS | COUNT | 22 | 20 | 42 |
| | % WITHIN GROUP | 44.0% | 40.0% | 42.0% |
| | % OF TOTAL | 22.0% | 20.0% | 42.0% |
| 30-40 YRS | COUNT | 22 | 28 | 50 |
| | % WITHIN GROUP | 44.0% | 56.0% | 50.0% |
| | % OF TOTAL | 22.0% | 28.0% | 50.0% |
| 40-50 YRS | COUNT | 2 | 0 | 2 |
| | % WITHIN GROUP | 4.0% | .0% | 2.0% |
| | % OF TOTAL | 2.0% | .0% | 2.0% |
| 50-60 YRS | COUNT | 3 | 1 | 4 |
| | % WITHIN GROUP | 6.0% | 2.0% | 4.0% |
| | % OF TOTAL | 3.0% | 1.0% | 4.0% |
| TOTAL | COUNT | 50 | 50 | 100 |
| | % WITHIN GROUP | 100.0% | 100.0% | 100.0% |
| | % OF TOTAL | 50.0% | 50.0% | 100.0% |

Chi Square Test: p Value=0.432

Most of the Hypothyroidism patients were in 20 t0 30 years of age distribution (44%) and most of the hyperthyroidism patients were in 30 to 40 years of age distribution (56%).

**Table 7. Sex distribution of patients with PHT in Hypothyroidism**

| PHT | | Sex | | Total |
|---|---|---|---|---|
| | | F | M | |
| Absent | Count | 29 | 12 | 41 |
| | % within Sex | 80.6% | 85.7% | 82.0% |
| | % of Total | 58.0% | 24.0% | 82.0% |
| Present | Count | 7 | 2 | 9 |
| | % within Sex | 19.4% | 14.3% | 18.0% |

Out of 9 patients with PHT in hypothyroidism group 7 females and 2 male patients were present. This gives 19.4% of females and 14.3% of males within sex.

**Table 8. Sex Distribution of Pericardial Effusion in patients with Hypothyroidism**

| PERICARDIAL EFFUSION | | SEX | | TOTAL |
|---|---|---|---|---|
| | | F | M | |
| ABSENT | COUNT | 29 | 12 | 41 |
| | % WITHIN SEX | 80.6% | 85.7% | 82.0% |
| | % OF TOTAL | 58.0% | 24.0% | 82.0% |
| MILD EFFUSION | COUNT | 2 | 0 | 2 |
| | % WITHIN SEX | 5.6% | .0% | 4.0% |
| | % OF TOTAL | 4.0% | .0% | 4.0% |
| MODERATE EFFUSION | COUNT | 5 | 2 | 7 |
| | % WITHIN SEX | 13.9% | 14.3% | 14.0% |
| | % OF TOTAL | 10.0% | 4.0% | 14.0% |
| TOTAL | COUNT | 36 | 14 | 50 |
| | % WITHIN SEX | 100.0% | 100.0% | 100.0% |
| | % OF TOTAL | 72.0% | 28.0% | 100.0% |

A. GROUP = HYPOTHYROIDISM

Chi Sq Test: p Value=0.666

Out of 50 patients in hypothyroidism group 9 patients had pericardial effusion. 7 of them had moderate effusion. 2 of them had mild effusion. Sex distribution wise 5.6% females had mild effusion.13.9% of female patients and 14.3% of male patients had moderate effusion within the sex distribution.

**Table 9. Sex distribution of MVP in hyperthyroidism group**

| MVP | | Sex | | Total |
|---|---|---|---|---|
| | | F | M | |
| Absent | Count | 34 | 9 | 43 |
| | % within Sex | 89.5% | 75.0% | 86.0% |
| | % of Total | 68.0% | 18.0% | 86.0% |
| present | Count | 4 | 3 | 7 |
| | % within Sex | 10.5% | 25.0% | 14.0% |
| | % of Total | 8.0% | 6.0% | 14.0% |
| Total | Count | 38 | 12 | 50 |
| | % within Sex | 100.0% | 100.0% | 100.0% |
| | % of Total | 76.0% | 24.0% | 100.0% |

a. Group = Hyperthyroidism

Chi Sq Test: p Value=0.337

Out of 7 patients among 50 hyperthyroidism patients had MVP. Hence 25% of male sex distribution was noticed with a female sex distribution of 10.5%.

**Table 10. Correlation various variables with varying T4 level in hypothyroidism**

| | | T4 level | LVEF% | IVS(s) | IVS(d) | LV(s) | LV mass |
|---|---|---|---|---|---|---|---|
| T4 level | Rho | 1.000 | -.010 | -.229 | -.190 | -.048 | .055 |
| | Sig. | . | .944 | .110 | .187 | .741 | .703 |
| | N | 50 | 50 | 50 | 50 | 50 | 50 |
| LVEF% | Rho | -.010 | 1.000 | -.386** | -.404** | -.374** | -.195 |
| | Sig. | .944 | . | .006 | .004 | .007 | .174 |
| | N | 50 | 50 | 50 | 50 | 50 | 50 |
| IVS(s) | Rho | -.229 | -.386** | 1.000 | **.976**** | **.555**** | .305* |
| | Sig. | .110 | .006 | . | .000 | .000 | .031 |
| | N | 50 | 50 | 50 | 50 | 50 | 50 |
| IVS(d) | Rho | -.190 | -.404** | .976** | 1.000 | .556** | .340* |
| | Sig. | .187 | .004 | .000 | . | .000 | .016 |
| | N | 50 | 50 | 50 | 50 | 50 | 50 |
| | Sig. | .741 | .007 | .000 | .000 | . | .081 |
| | N | 50 | 50 | 50 | 50 | 50 | 50 |
| | Sig. | .112 | .096 | .003 | .005 | .001 | .224 |
| | N | 50 | 50 | 50 | 50 | 50 | 50 |
| LVID(s) | Rho | .000 | -.211 | .431** | .412** | .416** | .229 |
| | Sig. | .999 | .142 | .002 | .003 | .003 | .109 |
| | N | 50 | 50 | 50 | 50 | 50 | 50 |
| LVID(d) | Rho | -.271 | -.201 | .361** | .343* | .549** | .042 |
| | Sig. | .057 | .161 | .010 | .015 | .000 | .773 |
| | N | 50 | 50 | 50 | 50 | 50 | 50 |
| LV mass index | Rho | .055 | -.195 | .305* | .340* | .249 | 1.000 |
| | Sig. | .703 | .174 | .031 | .016 | .081 | . |
| | N | 50 | 50 | 50 | 50 | 50 | 50 |

No significant correlation had been found with varying T4 (below or above a threshold level to designate hypo or hyperthyroidism) and other variables.

**Table 11. Correlation of T4 levels with DCM**

| | | | T4 level | Dilated Cardiomyopathy |
|---|---|---|---|---|
| Kendall's tau_b | T4 level | Correlation Coefficient | 1.000 | -.081 |
| | | Sig. (2-tailed) | . | .495 |
| | | N | 50 | 50 |
| | Dilated Cardiomyopathy | Correlation Coefficient | -.081 | 1.000 |
| | | Sig. (2-tailed) | .495 | . |
| | | N | 50 | 50 |

No significant correlation had been found between DCM and varying T4 below a lower level in hypothyroidism.

**Table 12. Correlation of T4 levelos with Pericardial Effusion**

| | | | T4 level | Pericardial Effusion |
|---|---|---|---|---|
| Kendall's tau_b | T4 level | Correlation Coefficient | 1.000 | -.062 |
| | | Sig. (2-tailed) | . | .597 |
| | | N | 50 | 50 |
| | Pericardial Effusion | Correlation Coefficient | -.062 | 1.000 |
| | | Sig. (2-tailed) | .597 | . |
| | | N | 50 | 50 |

No significant correlation found between T4 level and the grading of pericardial effusion after a lower T4 in hypothyroidism

**Table 13. Sex distribution of variables within hypothyroid group**

| | Sex | N | Mean | Std. Deviation | Std. Error Mean | T Test (p Value) |
|---|---|---|---|---|---|---|
| IVS(s) | Female | 38 | .8868 | .13788 | .02237 | 0.43 |
| | Male | 12 | .85 | .14460 | .04174 | |
| LVEF% | Female | 38 | 63.00 | 2.84747 | .46192 | 0.540 |
| | Male | 12 | 63.5000 | 2.27636 | .65713 | |
| IVS(d) | Female | 38 | .7237 | .14966 | .02428 | 0.268 |
| | Male | 12 | .6667 | .16697 | .04820 | |
| LV mass index | Female | 38 | 111.4211 | 24.13658 | 3.91547 | 0.983 |
| | Male | 12 | 111.6667 | 57.67674 | 16.64984 | |

The p value was insignificant in the test, so the sex distribution among the variables shown happened to be insignificant. Hence, there was no sex dependent alteration of LVEF, IVS(s), IVS (d) or LV mass in hypothyroidism.

**Table 14. Distribution of variables according to age in hypothyroidism group**

| | | Sum of Squares | df | Mean Square | F | Sig. |
|---|---|---|---|---|---|---|
| LVEF% | Between Groups | 741.390 | 4 | 185.347 | 1.405 | .248 |
| | Within Groups | 5935.030 | 45 | 131.890 | | |
| | Total | 6676.420 | 49 | | | |
| IVS(s) | Between Groups | .156 | 4 | .039 | 1.019 | .408 |
| | Within Groups | 1.718 | 45 | .038 | | |
| | Total | 1.874 | 49 | | | |
| IVS(d) | Between Groups | .156 | 4 | .039 | .921 | .460 |
| | Within Groups | 1.905 | 45 | .042 | | |
| | Total | 2.061 | 49 | | | |
| LV mass index | Between Groups | 10200.517 | 4 | 2550.129 | 1.264 | .298 |
| | Within Groups | 90777.803 | 45 | 2017.285 | | |
| | Total | 100978.320 | 49 | | | |

a. Group = Hypothyroidism

The p value was insignificant. So, there was actually no age distribution dependent variation in these variables. Hence, LVEF, IVS(s), IVS (d) or LV mass did not vary with variation of age in hypothyroidism in our test.

# 6. DISCUSSION

During a period of six months over 50 hypothyroid patients and 50 hyperthyroid patients from the Department of Endocrinology and Department of Medicine had been studied with 2D TTE.

Parameters like LVEF, LV mass, IVS(s), IVS(d), presence or absence of pericardial effusion, presence or absence of DCM and presence or absence of MVP were taken into consideration.

Patients were screened for Hypertension, CAD, DM, Valvular Heart Disease and COPD and excluded from the study. The results were carefully analyzed using EPI INFO 2002, designed by CDC, Atlanta.

Whether these parameters were changing with changing levels of T4 after a threshold level in hypothyroidism or hyperthyroidism had been analyzed.

Whether these parameters were changing with age and sex distribution in both hypothyroidism and hyperthyroidism group also had been analyzed as follows:

**1**. Nine out of 50 patients in Hypothyroidism group had DCM. None had DCM in the hyperthyroidism group. Hence the prevalence of DCM in the hypothyroidism group was 18%.

Dilated Cardiomyopathy in hypothyroidism had been studied in many International and Indian studies. Asymmetrical septal hypertrophy had been noticed in so many cases with hypothyroid cardiomyopathy. In a study by **Reza and AS Abbasi** 34% of hypothyroid individuals had DCM **(MJ Reza, AS Abbasi west J Med. 1975 sep 123(3) 228-230).**

The incidence as well as the severity of cardiomyopathy was correlated with T4levels in that study by **Reza et al** and found to have an inverse relationship.

In our study no such relationship between T4 level and severity of DCM had been noticed.

**2**. Seven out of 50 patients in Hyperthyroidism group had MVP. The prevalence was 14% within the group (Hyperthyroidism). Only one patient in the hypothyroidism group had MVP, hence the prevalence was 2% within the hypothyroidism group.

The prevalence of MVP in hyperthyroidism had been established by many studies.

**Channik et al** observed that the prevalence of MVP was 41% among 39 hyperthyroid cases studied. This was the first study which established a relationship between MVP and graves disease.

In a case control study by **Alexander bruman et al (Pub. Br. Heart J. 1985 53:374-7)** a prevalence of 16.3% for MVP found in hyperthyroid patients.

MVP used to be one of the feature of Hyperthyroidism. (51)

The association between MVP and Hyperthyroidism had been observed especially in Hashimoto's thyroiditis. (52)

**3.** Nine out of 50 patients in Hypothyroidism group had PHT. Hence the prevalence of PHT in hypothyroidism was 18%. None in hyperthyroidism group had PHT.

But in a study by **Hassan M.Ismail (J.Gen.Int.Medicine 2007 Jan 22(1) 148-150),** the prevalence of PHT was higher with hyperthyroidism than hypothyroidism.

**4.** Nine out of 50 patients with hypothyroidism had pericardial effusion. Hence the prevalence of pericardial effusion in the hypothyroidism group was 18%. That was very significant. In those patients with pericardial effusion 14% had moderate effusion within the group and 4% had mild effusion.

But no one had massive effusion or tamponade in our study, But in a case control study done by **MJ Reza et al** 30% to 80% of hypothyroidism patients had pericardial effusion and up to 50% of them developed tamponade.

**5.** Most of the Hypothyroidism patients were found in the 20 to 40 years age distribution (44%), and most of the hyperthyroidism patients were found in the 30 to 40 years age distribution (56%).

6. Out of 50 patients in the hypothyroidism group 9 patients had PHT and the prevalence was 18% for PHT in hypothyroidism group. Among them 7 were females (19.4% within sex distribution). Predominant female sex distribution for PHT in hypothyroidism was observed.

7. Out of 50 patients in the hypothyroidism group, 9 had pericardial effusion. Seven of them had moderate effusion and 2 of them had mild effusion. Sex distribution wise 5.6% female patients had mild effusion and 13.9% of female patients 14.3% of male patients had moderate effusion. Though pericardial effusion was predominant among female patients, male patients were having more severe effusion than female patients. A study by **Reza et al** concluded that there was an observed 1.4:1 female preponderance in

developing pericardial effusion in hypothyroidism but the ratio used to vary in different studies.

**8**. No significant correlation had been found between varying T4 level (below or above a level to designate hypo or hyperthyroidism) and other parameters.

Hence the T4 level and development of complications happened to be an all or none phenomenon. **Rawat B et al** also observed that there was no significant difference in various diameters among different groups in hypothyroidism in a case control study

But in most of the International studies, we had come across a definite relationship between the variation of the T4 level and severity of DCM, septal thickness, hypokinesia etc., May be that was due to the prolonged latent period between the onset of deficiency to detection of cardiovascular abnormalities and the linear relationship with time might had been misinterpreted. More over our study population were fresh cases rather than treated or partially treated cases.

**9**. No significant correlation had been found between DCM and varying T4 level, below a lower level in hypothyroidism. So, once the Thyroxine started decreasing the adverse cardiovascular effects were ignited and started smoldering unless Thyroxine reintroduced. As we construed above that was

due to increased interval between onset of the deficiency to the overt clinical symptoms or screening interval which might have influenced the severity of the lesion rather than the varying hormone level (below a lower level).

**10**. No significant correlation found between T4 level and the grading of pericardial effusion after a lower T4 level in hypothyroidism. The all or none phenomena of T4 level could be considered here also.

So, in our study in the Hypothyroidism group, 18% of patients had Pericardial Effusion and 18% had Dilated Cardiomyopathy. They were all symptomatic.

Female patients out number male patients in developing complications in Hypothyroidism.

Patients with Hyperthyroidism had MVP with grade I or II MR, they were asymptomatic in our study and found incidentally.

**11**. The p value was insignificant while testing for the distribution of sex dispersion in LVEF, IVS(s), IVS (d) or LV mass in hypothyroidism. Hence there was no sex dependent alteration of LVEF, IVS(s), IVS (d) or LV mass in hypothyroidism.

But anyway the incidence of Hypothyroidism/Hyperthyroidism and development of complications were prevalent in female patients when compare to male patients.

**12. T**he p value was insignificant while testing the age distribution in LVEF, IVS (s), IVS (d) or LV mass. So, there was actually no age distribution dependent variation in these variables. Hence, LVEF, IVS(s), IVS (d) or LV mass did not vary with variation of age (in hypothyroidism group )in our test. In a study from Kathmandu by **Rawal B and Satyal A (Kathmandu University Medical Journal 2003 vol.2, No.3, issue 7, 182-187)** and in a study by **Bernnet et al (1983), Lee et al(1990) and Bernstein et al (1995)** concluded that there was increased septal thickness in elderly and it was due to age related thickness of the IVS superimposed by the changes induced by hypothyroidism.

# 7. CONCLUSION

- From our study we have come to know that significant percentage of patients with hypothyroidism developed various cardiovascular morbidities like DCM and pericardial effusion, moreover the level of T4 below a lower threshold level in hypothyroidism does not influence the severity of DCM or pericardial effusion or chamber size.
- Out of 50 hypothyroid patients 9 patients had DCM and 9 patients had pericardial effusion. The patients with DCM had moderate to severe LV dysfunction. They were all symptomatic.
- Out of 50 hyperthyroid patients 7 patients had MVP with mild MR. they were asymptomatic and detected incidentally during screening.
- No age related increase in severity of LV dysfunction noticed in our study. The main influence was by the hypothyroid status per se.
- The study outcome emphasized the importance of non ischemic causes like hypothyroidism in causing DCM and LV dysfunction.
- The outcome made us to think about the sub clinical hypothyroidism where the overt clinical manifestations would not occur and the underlying cardiac malfunctions started smoldering.

- As most of the cardiac morbidities are reversible after treatment, one has to screen for thyroid status of the patient while evaluating cases of DCM, pericardial effusion and MVP to institute an early rescue.
- Apart from these anatomical and functional alterations, hypothyroidism increases the likelihood of CAD through its pro atherogenic effect.
- So, early detection and treatment of thyroid deficiency plays a vital role in preventing this malfunctions to go to an irreversible state.
- Moreover hyperthyroidism is an important cause of non rheumatic atrialfibrillation.
- It is worthwhile to screen for thyroid status in patients with CAD and in all young patients with hypercholesterolemia.
- Otherwise, the prevalence of thyroid disorders in tropical countries like India act synergistically with other factors like urbanization and changing food habits and smoking, in increasing cardiovascular morbidity and mortality.
- Another point to ponder is the prevalence of hypothyroidism and its complications in female sex, made it mandatory to screen for thyroid deficiency in all women with CAD, pericardial effusion or cardiomyopathy.

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**A PROSPECTIVE ECHOCARDIOGRAPHIC STUDY IN PATIENTS WITH HYPOTHYROIDISM AND HYPERTHYROIDISM**

**Name:** **Age:** **Sex:**

**Address:**

**Symptoms:**
Palpitation Chest Pain Dyspnea NYHA class:

Fatigue Somnolence Cold/Heat intolerance Tremor Fever

Giddiness Syncope Goitre

**Co morbid Conditions:**

**Personal H/O:**

Smoker Alcoholic Exposure to STD

**Menstrual/Obstetric H/O:**

**General Examinations:**

Pulse BP Anemia Pedal edema

**CVS:**

JVP
S1 S2 Murmur

**Laboratory Analysis:**

Hb% TC DC- P L E M ESR

Blood Sugar Urea Sr.Creatinine

**T3** **T4** **TSH**

**ECG in all leads:**

**A PROSPECTIVE ECHOCARDIOGRAPHIC STUDY IN PATIENTS WITH HYPOTHYROIDISM AND HYPERTHYROIDISM**

## 2D ECHOCARDIOGRAM

LV function LVID(s) LVID(d) LVEF %

Stroke Volume

Wall Thickness IVS(s) IVS(d) LV(s) LV(d)

Pericardial Effusion Mild Moderate Severe

Wall Motion Abnormalities Hypokinesia Akinesia Dyskinesia

IV Paradoxical Movement a b c

| Valves | Stenosis | Regurgitation |
|---|---|---|
| Mitral | | |
| Tricuspid | Pulmonary | |
| Aortic | | |

Pulmonary HT TRPG

**Impression:**

# Abbreviations

IP no.- In patients Number

NYHA- New York Heart Association

TSH-Thyroid Stimulating Hormone

T4- Thyroxine

T3-Tri iodo thyronine

TTE- Trans Thoracic Echocardiography

RWMA-Regional Wall Motion Abnormalities

LVEF- Left Ventricular Ejection Fraction

LV mass-Left Ventricular Mass

IVSD-Inter Ventricular Septal Diameter

(s)-in systole, (d)- in diastole

PE-Pericardial Effusion

MR-Mitral Regurgitation

MVP-Mitral Valve Prolapse

PHT- Pulmonary Hypertension

PR-Pulmonary Regurgitation

TR-Tricuspid Regurgitation

DCM-Dilated Cardiomyopathy

AIIMS-All India Institute of Medical Sciences

**Figure1. Incidence of Cardiomyopathy in hypothyroidism and hyperthyroidism**

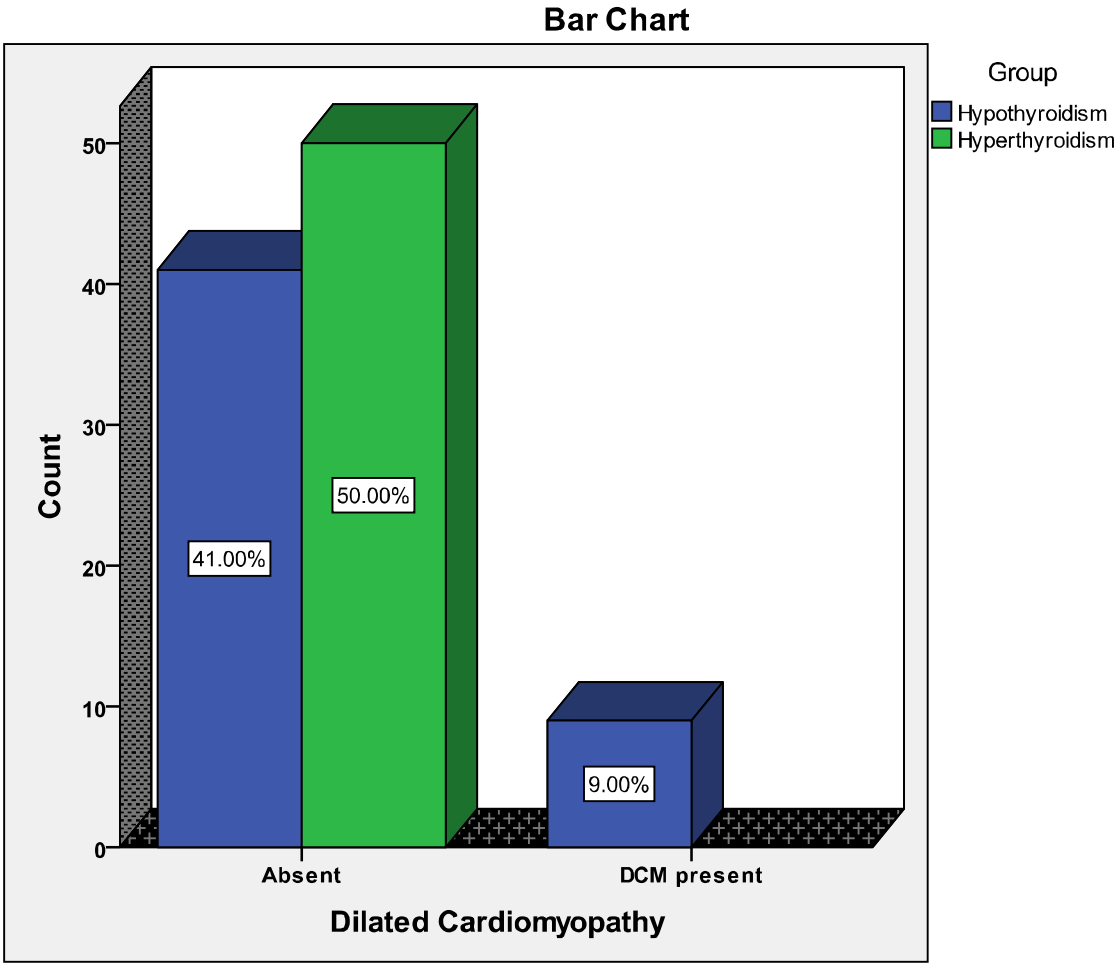


**Figure 2.Incidence of MVP in study groups**

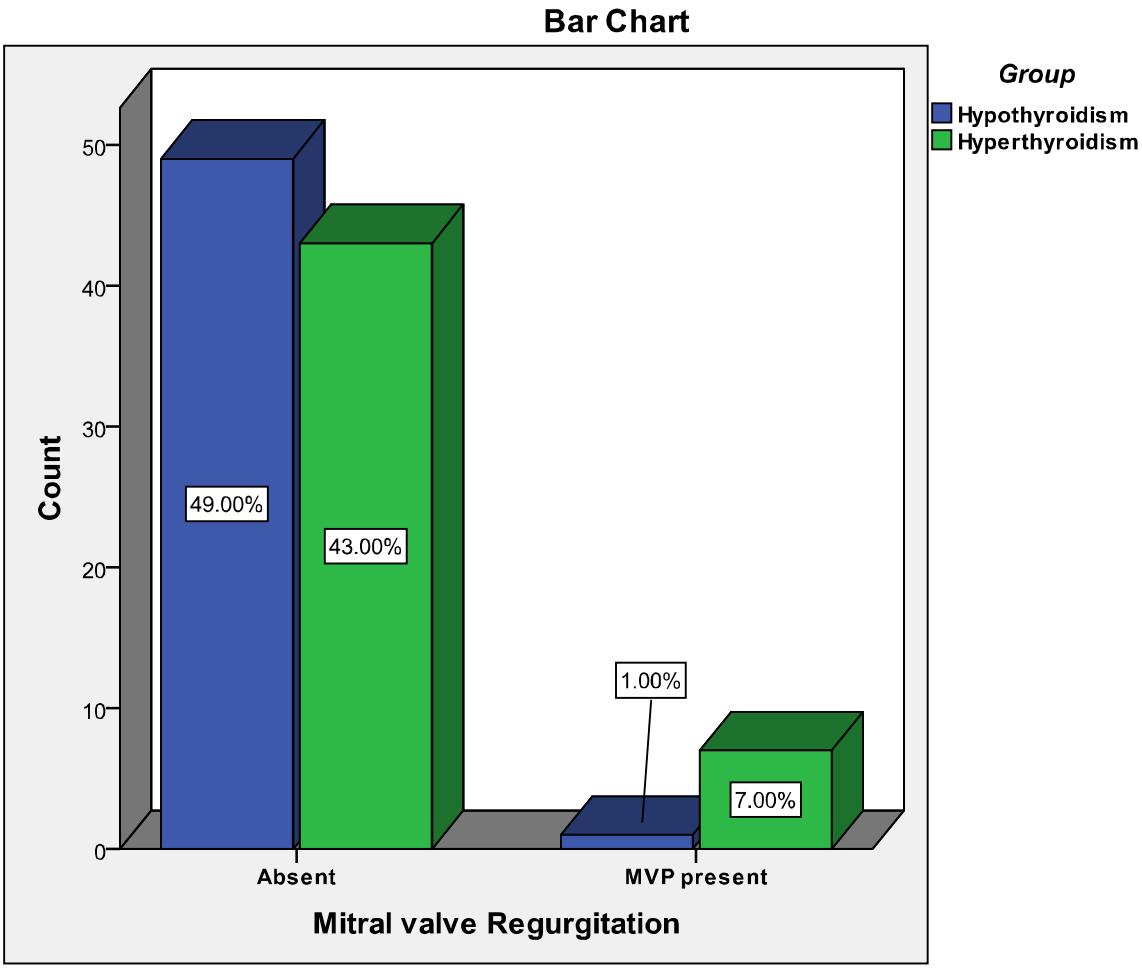


**Figure 3. Incidence of PHT in the study groups**

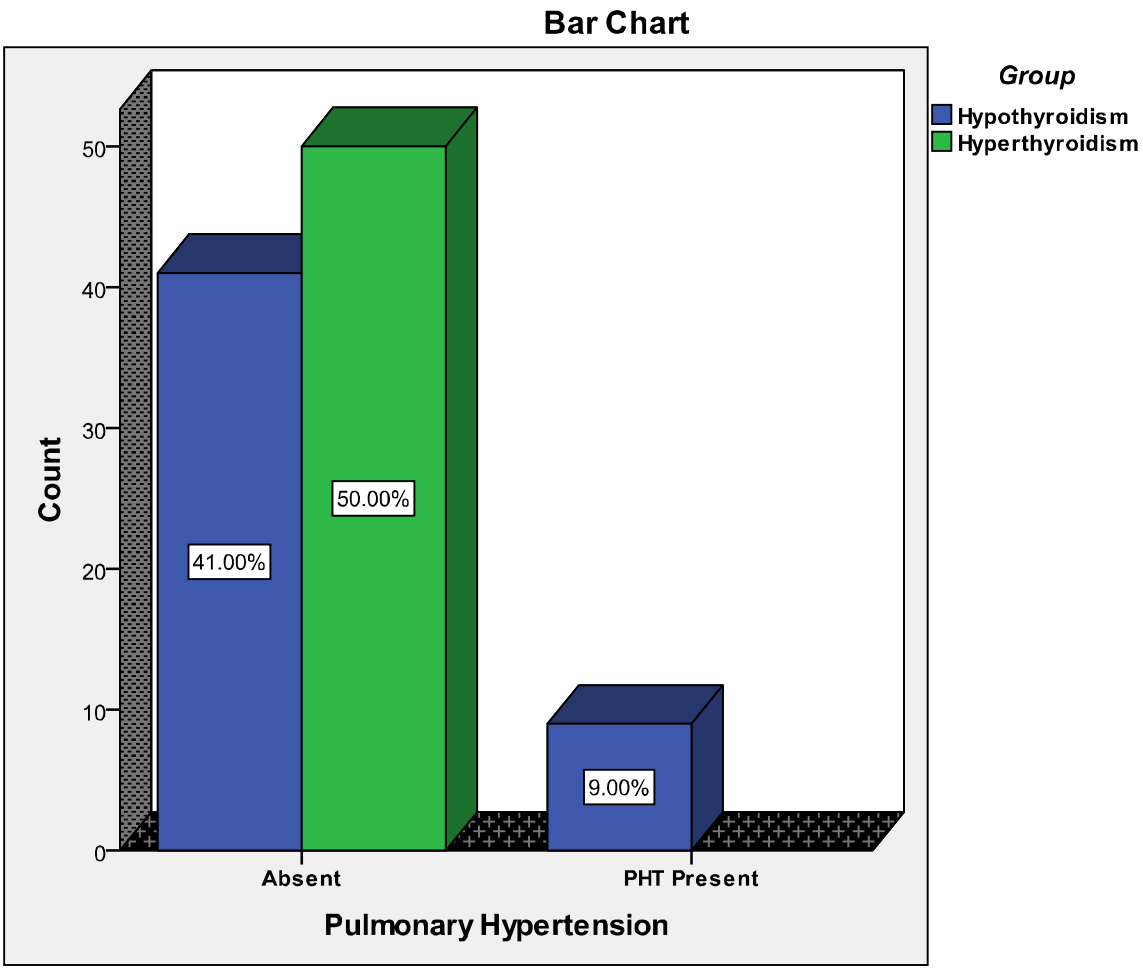


**Figure 4. Incidence of Pericardial Effusion in the study group**

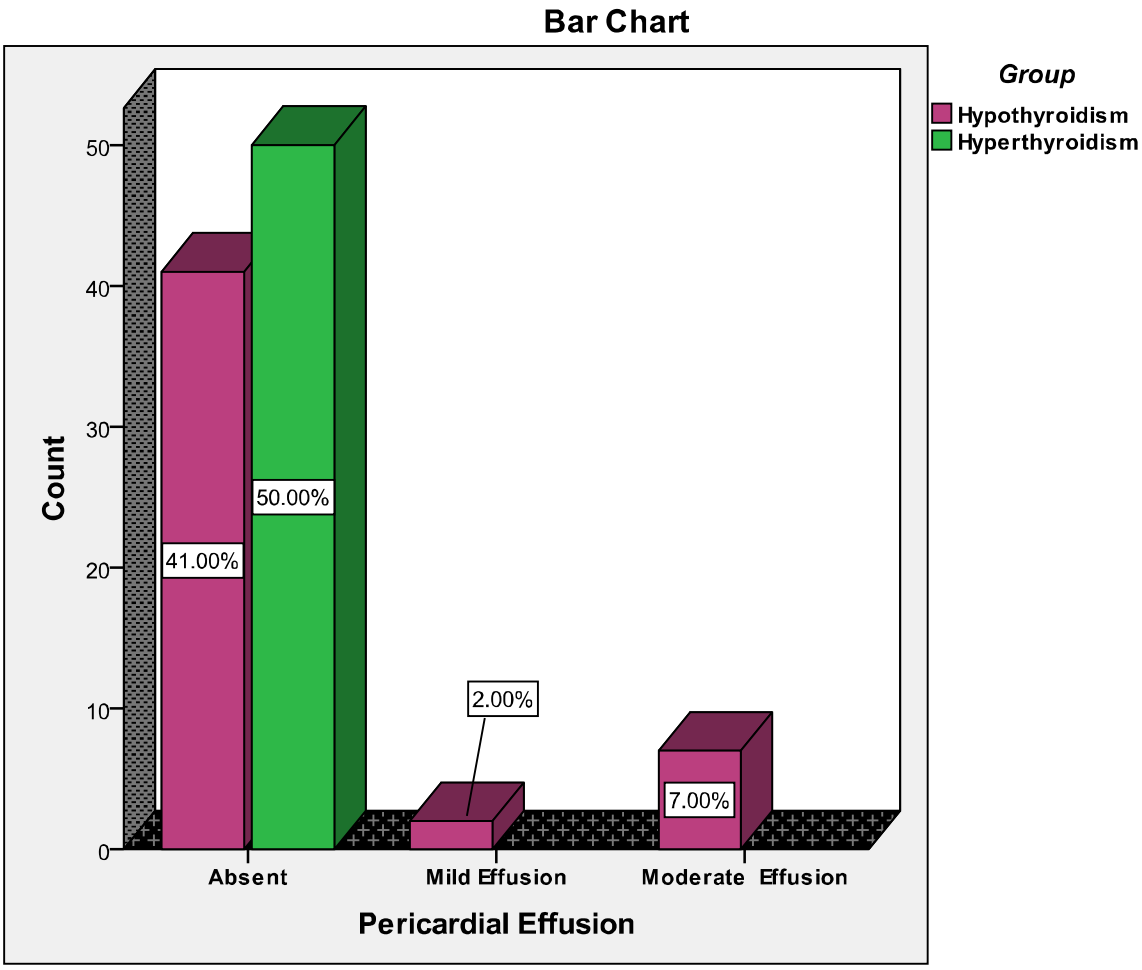


**Figure 5. Age distribution within hypothyroidism and hyperthyroidism group**

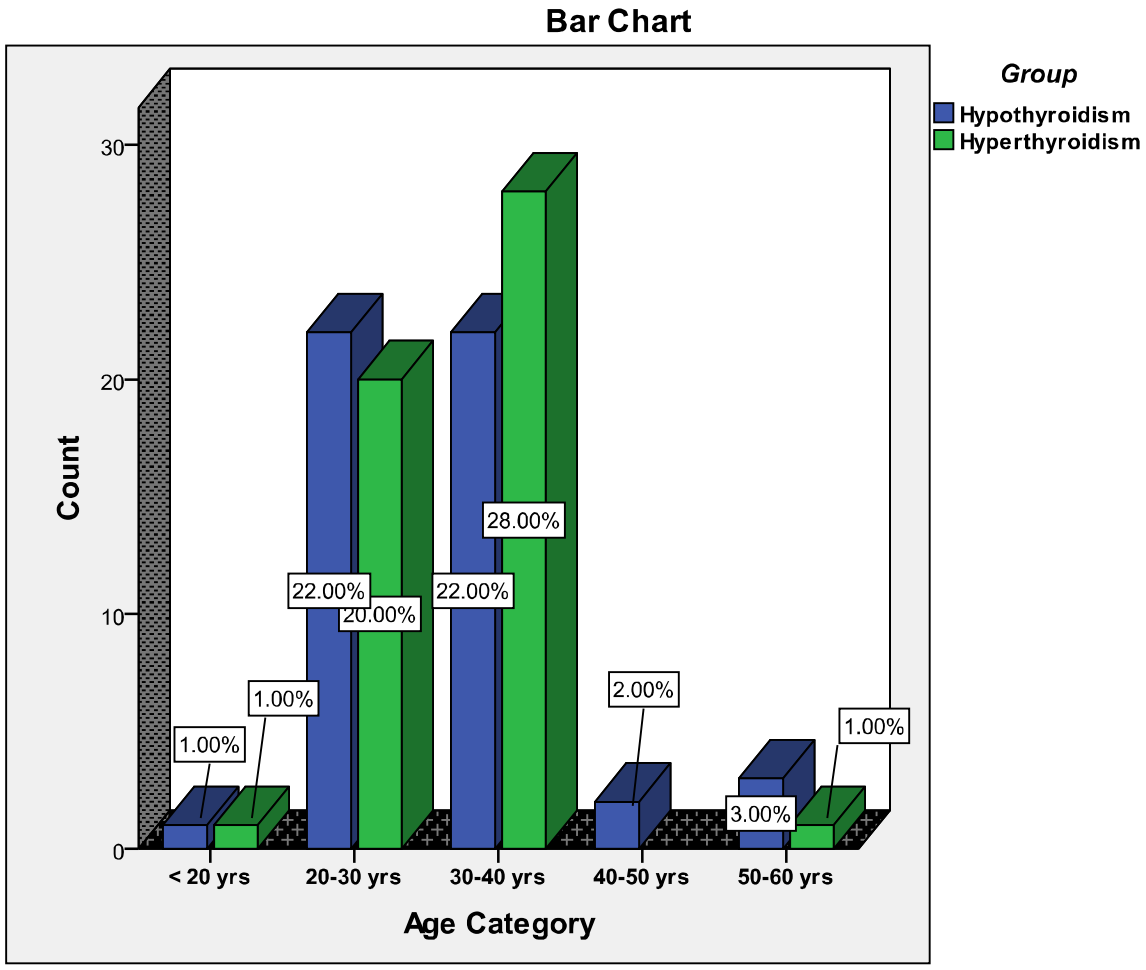


**Figure 6. Sex distribution of PHT in hypothyroidism**

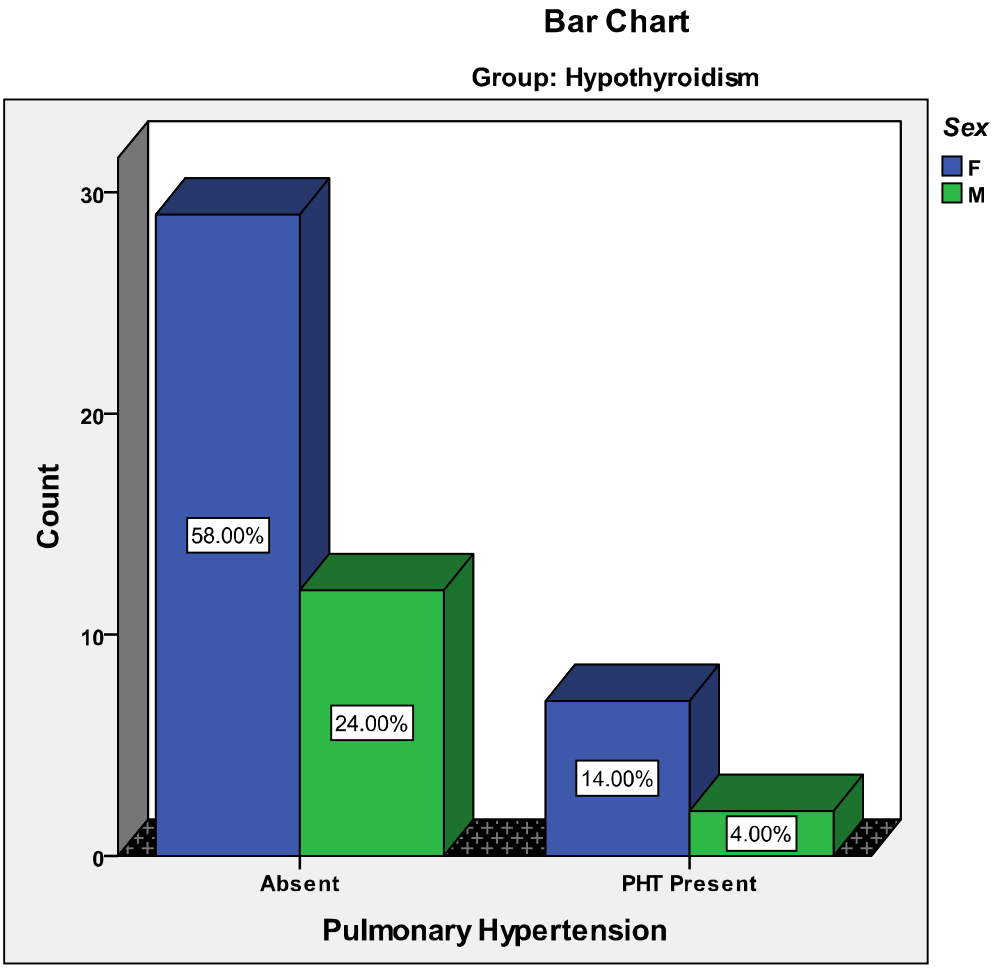


**Figure 7. Sex distribution of DCM in hypothyroidism**

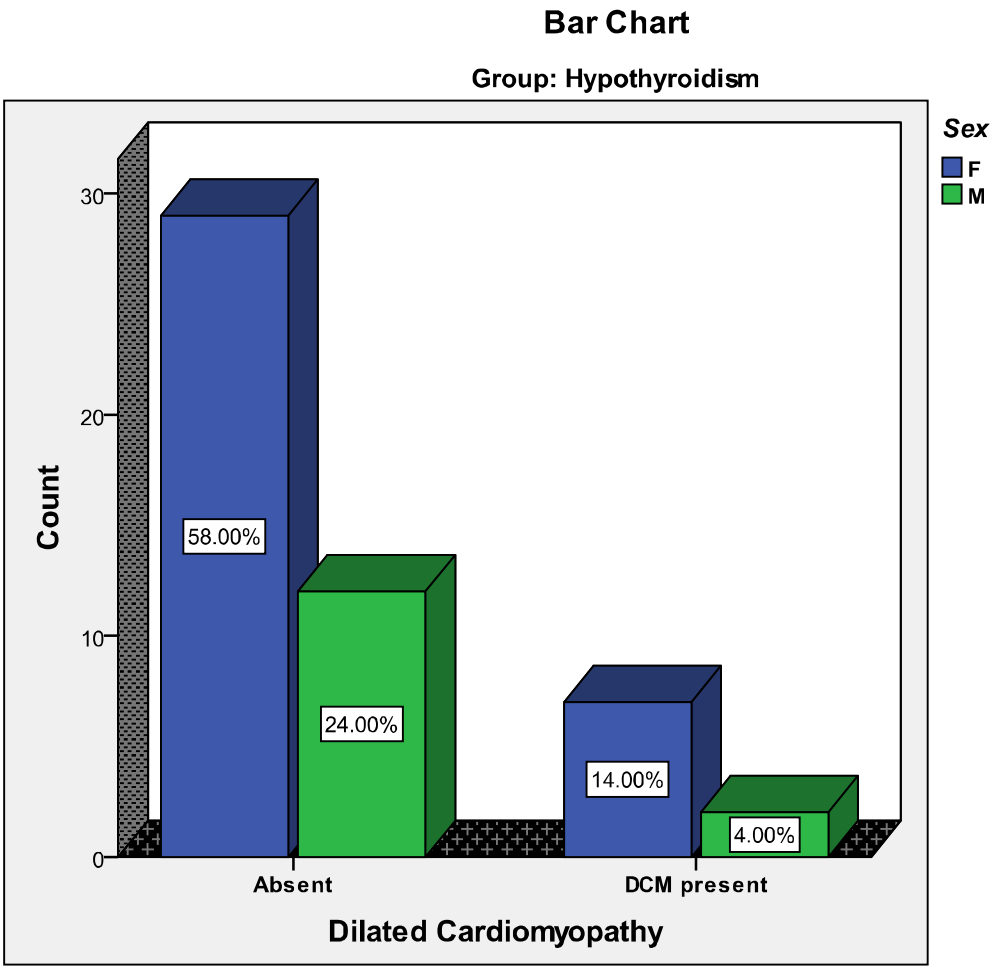


**Figure 8. Correlation of T4 levels with LV mass**

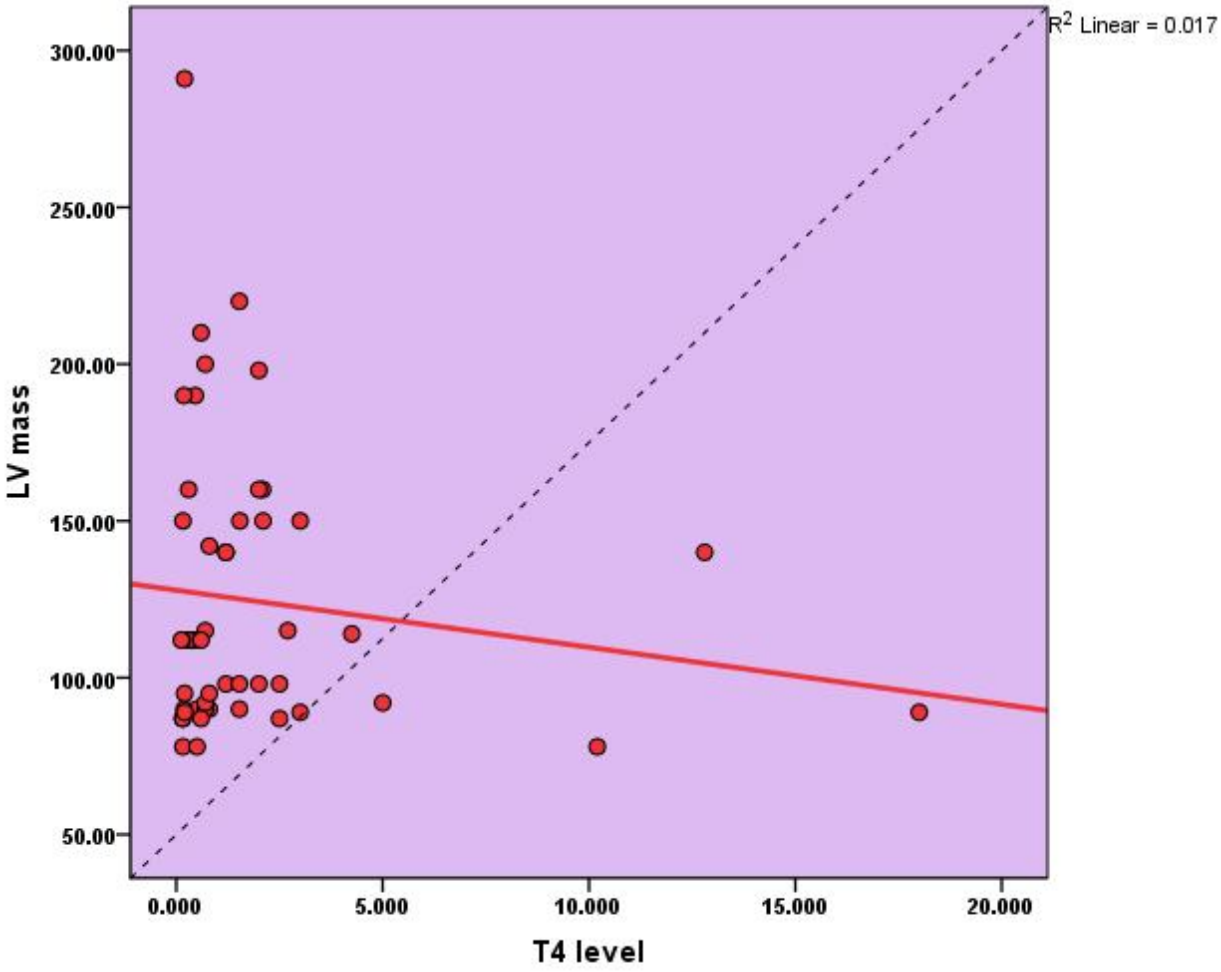


**Figure 9. Correlation of T4 level with Pericardial Effusion**

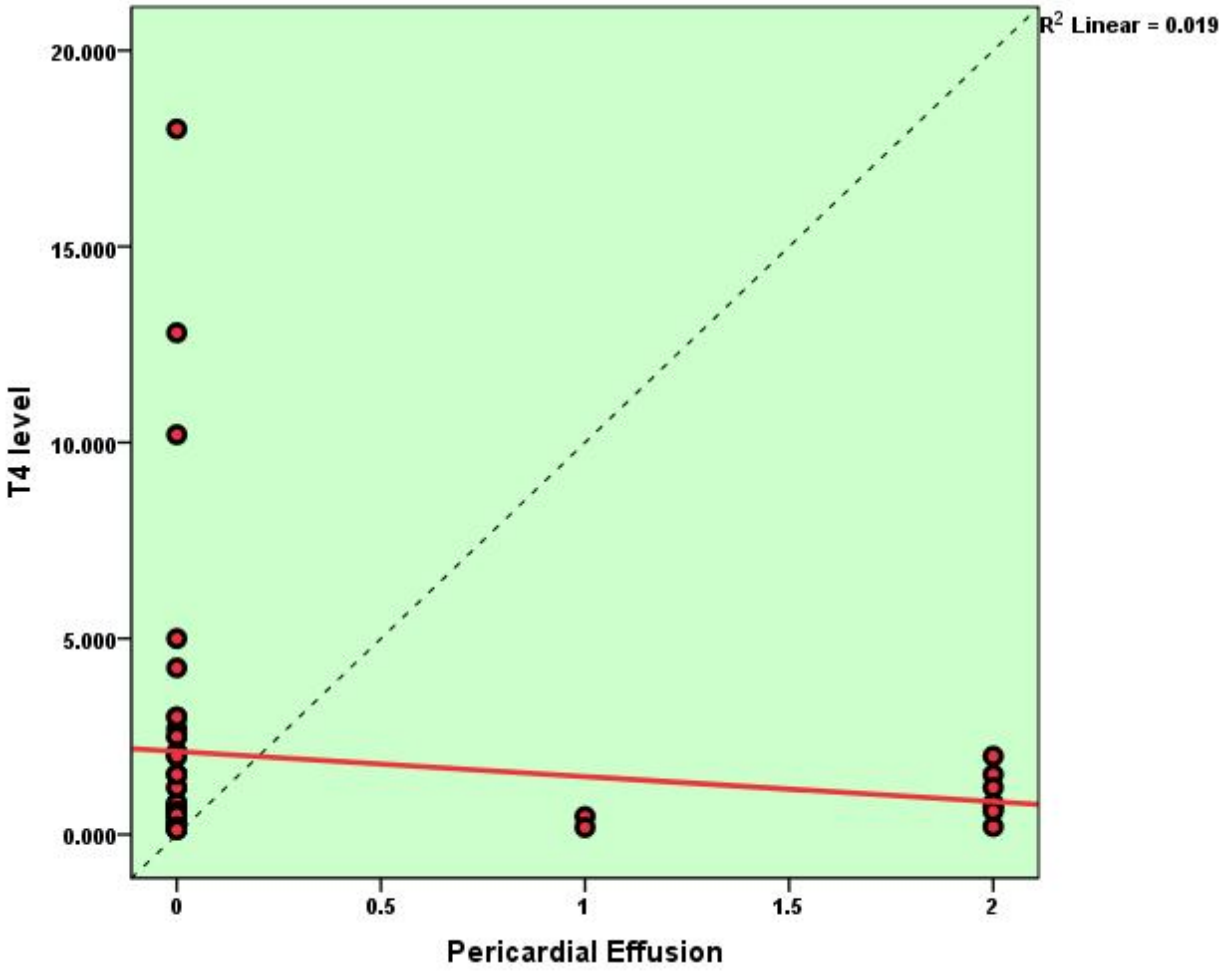


**Figure10. Correlation of T4 levels with DCM**

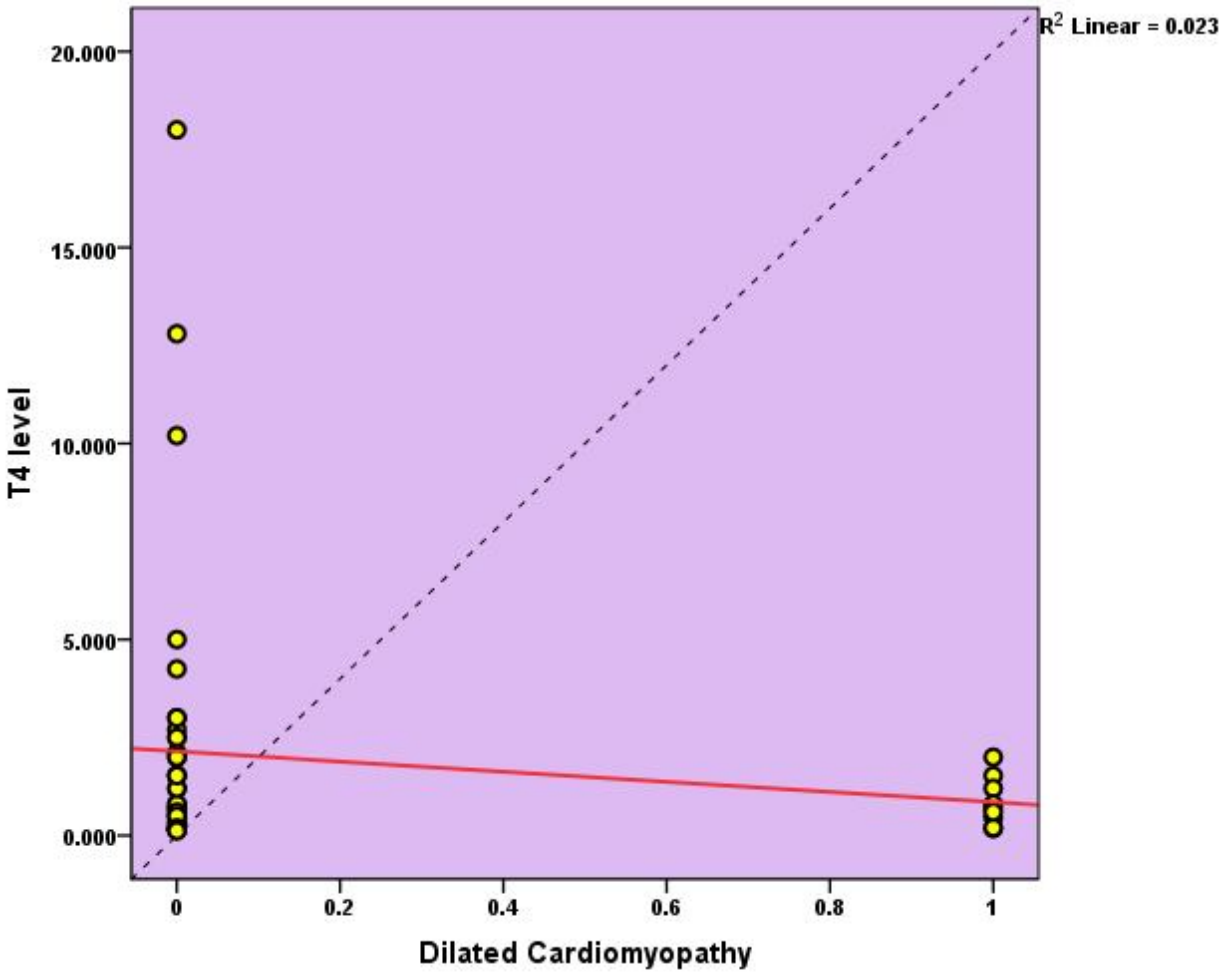
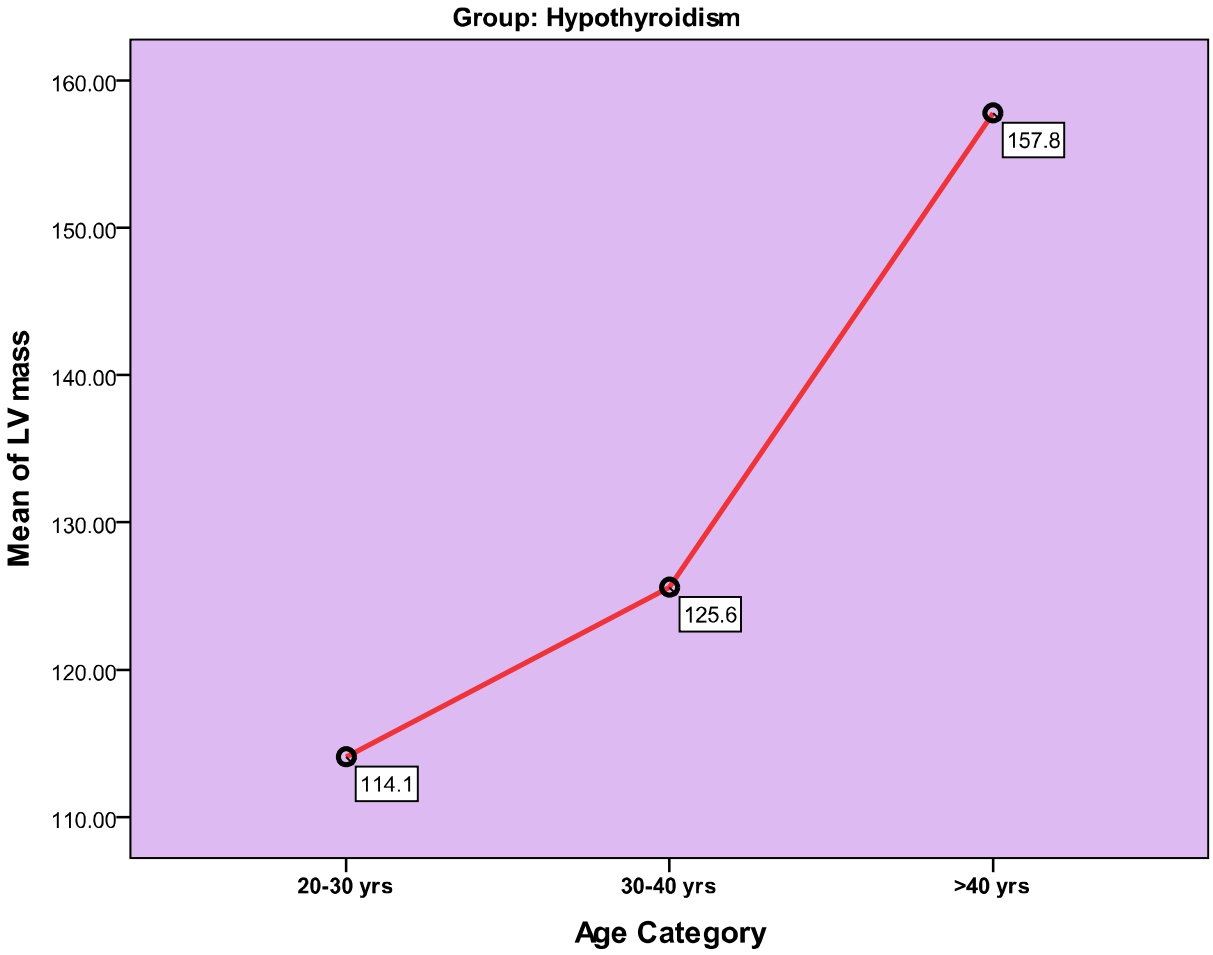
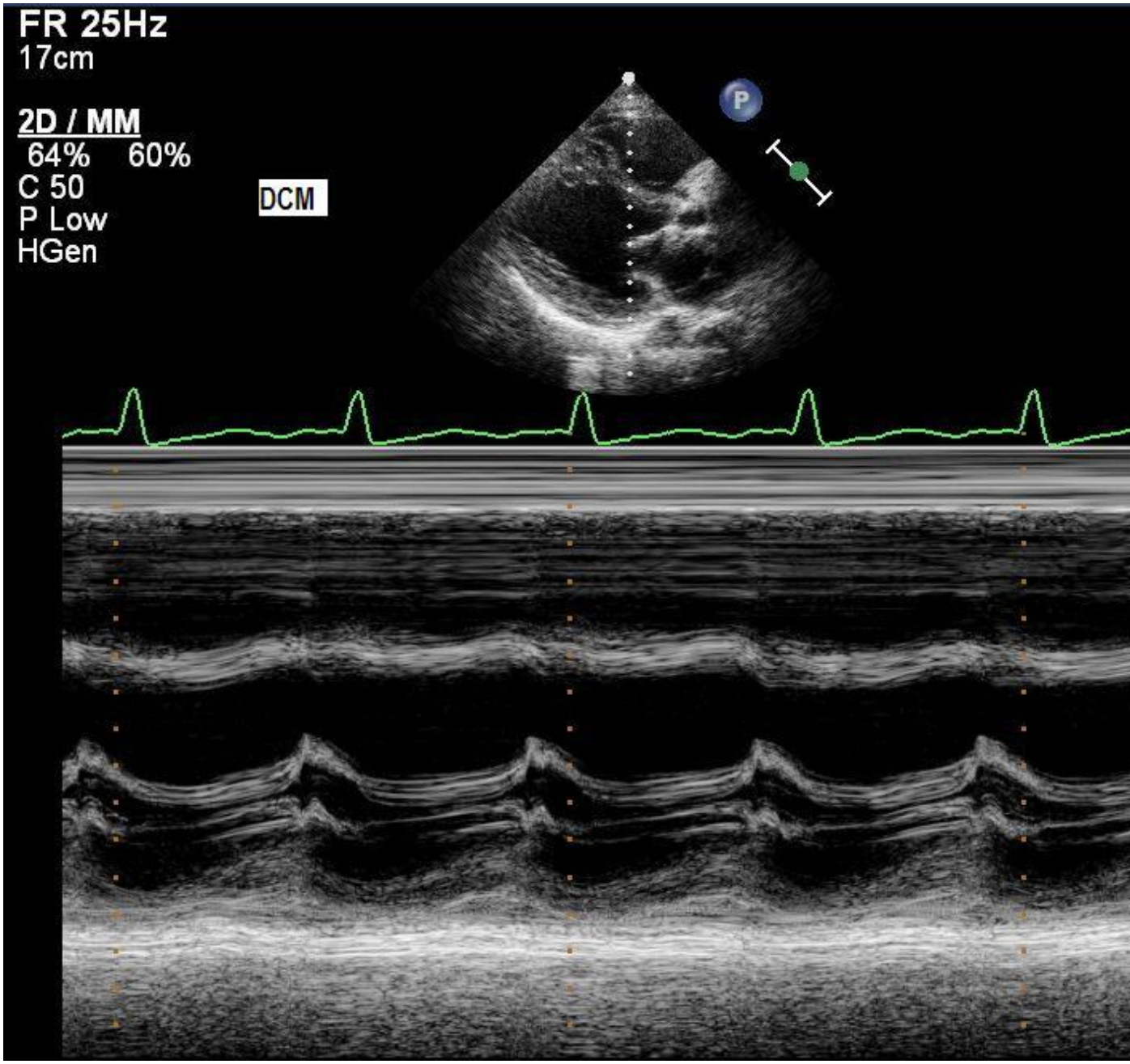


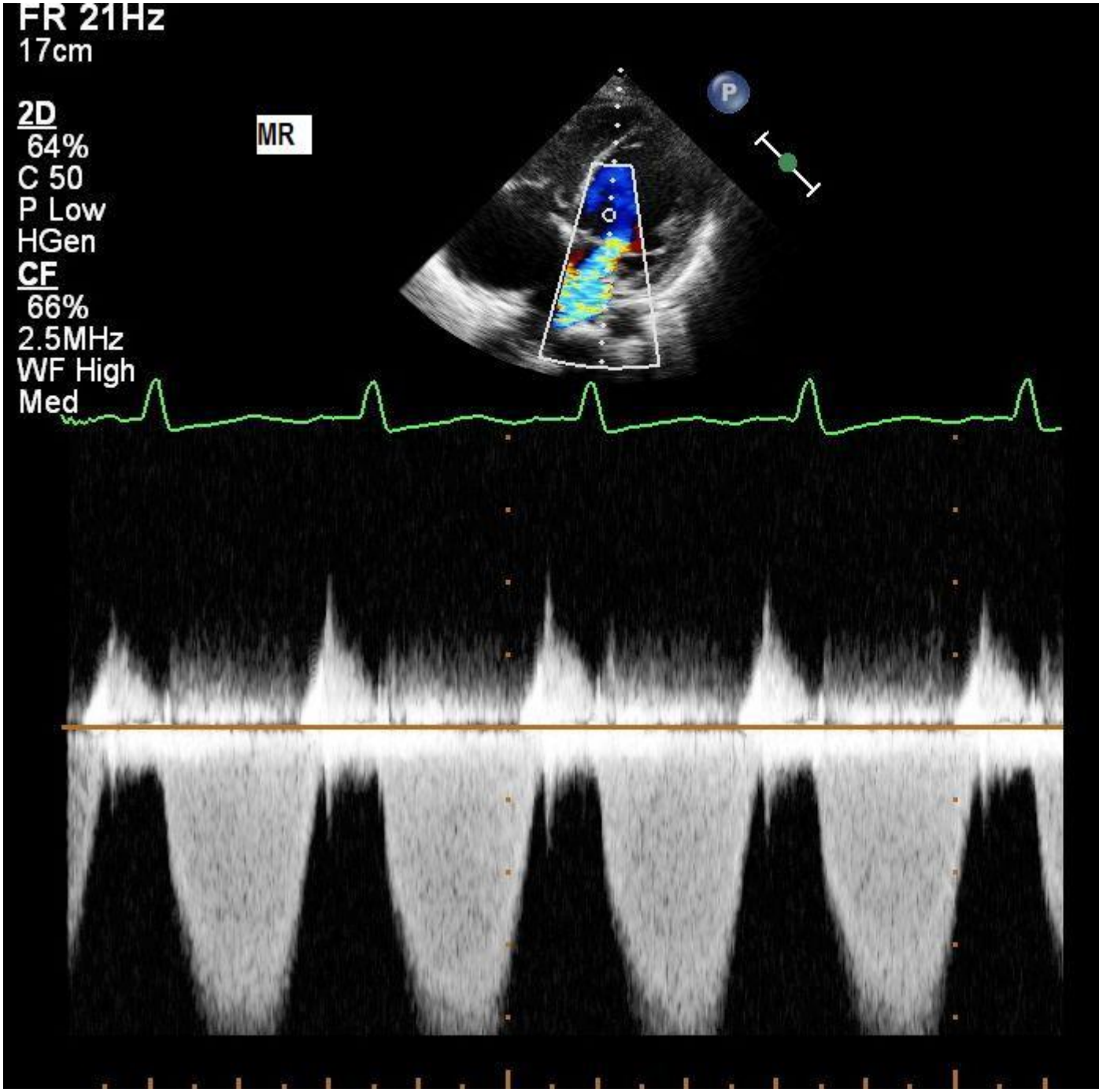
**Figure 11. Distribution of LV Mass according to age in hypothyroidism**



**Echo Picture 1. DCM in a hypothyroid female**



**Echo Picture 2.Mitral Regurgitation /DCM in a hypothyroid female**



# Master Chart

## Hypothyroidism Group

| S.No | IP No. | Age | Sex | T3 | T4 | TSH | Status | Pulse | B.P | NYHA | LVEF% | IVS(s) | IVS(d) | LV mass | RWMA | MR | MVP | TR | PR | PHT | TRPG | PE | DCM |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1 | 38978 | 22 | F | 21.6 | 0.4 | >150 | Hypo | 64 | 90/70 | II | 62 | 0.7 | 0.5 | 112 | No | No | No | No | No | No | NO | No | |
| 2 | 43567 | 36 | F | 24 | 0.46 | 100 | Hypo | 60 | 90/60 | III | 30 | 1.2 | 1 | 190 | Yes | Gr I | No | TR + | PR + | PHT + | 40 | Mild | + |
| 3 | 39545 | 32 | M | 22 | 1.53 | 62 | Hypo | 72 | 118/70 | I | 65 | 1.3 | 1.1 | 220 | Yes | MR + | No | TR + | PR + | PHT + | 35 | Mod | + |
| 4 | 38497 | 32 | M | 22 | 1.53 | 62 | Hypo | 60 | 110/70 | I | 60 | 1 | 0.9 | 90 | No | No | No | No | No | No | NO | No | |
| 5 | 41451 | 22 | M | 82 | 3 | >120 | Hypo | 70 | 100/70 | II | 60 | 0.8 | 0.6 | 89 | No | No | No | No | No | No | NO | No | |
| 6 | 45434 | 22 | F | 15.4 | <.70 | 65.4 | Hypo | 64 | 100/80 | II | 65 | 0.9 | 0.7 | 115 | No | No | No | No | No | No | NO | No | |
| 7 | 43654 | 34 | M | 54 | 1.2 | 88.7 | Hypo | 68 | 110/80 | II | 62 | 1 | 0.8 | 140 | No | No | No | No | No | No | NO | No | |
| 8 | 44234 | 32 | F | 18 | <.80 | >150 | Hypo | 60 | 90/70 | I | 65 | 0.9 | 0.7 | 90 | No | No | No | No | No | No | NO | No | |
| 9 | 45323 | 26 | M | 84 | 2.5 | >150 | Hypo | 70 | 100/70 | II | 60 | 0.9 | 0.7 | 87 | No | No | No | No | No | No | NO | No | |
| 10 | 43356 | 32 | F | 24 | 5 | 49.4 | Hypo | 70 | 120/90 | I | 67 | 0.7 | 0.5 | 92 | No | No | No | No | No | No | NO | No | |
| 11 | 47435 | 23 | F | 16.8 | <.50 | >100 | Hypo | 60 | 110/70 | I | 65 | 0.6 | 0.5 | 112 | No | No | No | No | No | No | NO | No | |
| 12 | 43434 | 53 | M | 34 | 2.09 | 20 | Hypo | 60 | 120/80 | II | 60 | 0.8 | 0.7 | 160 | No | No | No | No | No | No | NO | No | |
| 13 | 43335 | 30 | F | 45.3 | 0.16 | 114 | Hypo | 76 | 130/80 | I | 65 | 1 | 0.9 | 150 | No | No | No | No | No | No | NO | No | |
| 14 | 43543 | 40 | F | 20.3 | <.60 | >150 | Hypo | 58 | 90/60 | III | 35 | 1.1 | 1.3 | 210 | Yes | Gr II | | TR + | PR + | PHT + | 40 | Mod | + |
| 15 | 45232 | 54 | F | 50 | 0.18 | >100 | Hypo | 80 | 90/70 | III | 30 | 1.3 | 1.1 | 190 | Yes | Gr.II | No | TR+ | PR+ | PHT + | 30 | Mild | + |
| 16 | 45123 | 30 | F | 81 | 10.2 | 50 | Hypo | 64 | 110/70 | I | 60 | 1 | 0.9 | 78 | No | No | No | No | No | No | NO | No | |
| 17 | 42232 | 28 | M | 52 | 0.2 | >120 | Hypo | 62 | 90/70 | III | 25 | 0.9 | 0.7 | 291 | No | Gr II | No | TR + | PR+ | PHT+ | 35 | Mod | + |
| 18 | 34452 | 33 | M | 16.3 | <.30 | 78 | Hypo | 64 | 120/70 | I | 67 | 0.9 | 0.7 | 112 | No | No | No | No | No | No | NO | No | |
| 19 | 34542 | 32 | F | 0.2 | <15 | 110 | Hypo | 60 | 110/70 | II | 65 | 0.7 | 0.5 | 87 | No | No | No | No | No | No | NO | No | |
| 20 | 35123 | 34 | M | 15 | 4.25 | 99.5 | Hypo | 72 | 110/70 | II | 65 | 0.6 | 0.5 | 114 | No | No | No | No | No | No | NO | No | |
| 21 | 34654 | 30 | F | 0.1 | 0.5 | 150 | Hypo | 70 | 120/70 | II | 60 | 0.6 | 0.5 | 90 | No | No | No | No | No | No | NO | No | |
| 22 | 43435 | 36 | F | 22.5 | 18 | 48.4 | Hypo | 64 | 110/60 | I | 60 | 0.7 | 0.5 | 89 | No | No | No | No | No | No | NO | No | |
| 23 | 34453 | 30 | M | 20 | 2.7 | >100 | Hypo | 60 | 112/74 | II | 64 | 0.9 | 0.7 | 115 | No | No | No | No | No | No | NO | No | |
| 24 | 45233 | 34 | F | 40.4 | 1.2 | >150 | Hypo | 72 | 90/70 | III | 30 | 1 | 0.9 | 140 | No | Gr I | No | TR+ | PR+ | PHT + | 40 | Mod | + |
| 25 | 46544 | 24 | F | 0.2 | 0.7 | 100 | Hypo | 64 | 112/60 | II | 65 | 1 | 0.9 | 90 | No | No | No | No | No | No | NO | No | |

Hypoyhyroidism group contd…

| S.No | IP No. | Age | Sex | T3 | T4 | TSH | Status | Pulse | B.P | NYHA | LVEF% | IVS(s) | IVS(d) | LV mass | RWMA | MR | MVP | TR | PR | PHT | TRPG | PE | DCM |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 26 | 48456 | 25 | F | 53 | 0.15 | 62 | Hypo | 70 | 100/70 | I | 60 | 0.9 | 0.7 | 87 | No | No | No | No | No | No | NO | No | |
| 27 | 45567 | 26 | F | 17.3 | <.70 | 87.4 | Hypo | 64 | 100/70 | II | 60 | 0.9 | 0.7 | 92 | No | No | No | No | No | No | NO | No | |
| 28 | 45654 | 25 | F | 23.5 | 0.6 | 88.72 | Hypo | 74 | 110/80 | I | 65 | 0.9 | 0.7 | 112 | No | No | No | No | No | No | NO | No | |
| 29 | 47125 | 22 | F | 16 | 2 | 100 | Hypo | 60 | 120/80 | II | 62 | 0.9 | 0.7 | 160 | No | No | No | No | No | No | NO | No | |
| 30 | 42456 | 35 | F | 83 | 3 | >150 | Hypo | 64 | 100/80 | II | 65 | 0.7 | 0.5 | 150 | No | No | No | No | No | No | NO | No | |
| 31 | 43543 | 23 | F | 25 | 2.5 | >120 | Hypo | 68 | 120/70 | I | 60 | 0.6 | 0.5 | 98 | No | No | No | No | No | No | NO | No | |
| 32 | 45564 | 29 | F | 17 | 0.8 | 50 | Hypo | 60 | 90/70 | II | 67 | 0.6 | 0.5 | 95 | No | No | No | No | No | No | NO | No | |
| 33 | 43653 | 34 | M | 42 | 0.16 | >100 | Hypo | 70 | 100/70 | II | 65 | 0.7 | 0.5 | 78 | No | No | No | No | No | No | NO | No | |
| 34 | 45433 | 50 | F | 20 | <.70 | >150 | Hypo | 58 | 90/70 | III | 30 | 1.2 | 1 | 200 | Yes | MR + | No | TR + | PR + | PHT + | 40 | Mod | + |
| 35 | 36876 | 36 | F | 54 | 0.19 | 48.2 | Hypo | 70 | 120/70 | II | 65 | 1 | 0.9 | 90 | No | No | No | No | No | No | NO | No | |
| 36 | 37344 | 23 | F | 16.23 | <.60 | >100 | Hypo | 72 | 130/90 | II | 65 | 1 | 0.9 | 87 | No | No | No | No | No | No | NO | No | |
| 37 | 36452 | 33 | F | 54 | 2 | >150 | Hypo | 60 | 90/70 | III | 34 | 1.3 | 1.1 | 198 | No | Gr I MR | No | TR + | PR + | PHT + | 35 | Mod | + |
| 38 | 37356 | 25 | F | 14 | <.15 | 114 | Hypo | 64 | 100/70 | I | 60 | 0.9 | 0.7 | 112 | No | No | No | No | No | No | NO | No | |
| 39 | 35445 | 19 | F | 21 | <.30 | 100 | Hypo | 62 | 120/90 | II | 67 | 0.7 | 0.5 | 160 | No | No | No | No | No | No | NO | No | |
| 40 | 46322 | 34 | F | 23 | 1.54 | 62 | Hypo | 64 | 100/70 | II | 65 | 0.6 | 0.5 | 150 | No | No | No | No | No | No | NO | No | |
| 41 | 43535 | 29 | F | 35 | 1.2 | 80.2 | Hypo | 80 | 90/70 | I | 65 | 0.8 | 0.7 | 98 | No | No | No | No | No | No | NO | No | |
| 42 | 46335 | 35 | M | 25 | 0.2 | >110 | Hypo | 70 | 110/80 | I | 60 | 1 | 0.9 | 95 | No | No | No | No | No | No | NO | No | |
| 43 | 44564 | 38 | M | 16.4 | <.50 | >100 | Hypo | 76 | 130/80 | II | 60 | 0.8 | 0.6 | 78 | No | No | No | No | No | No | NO | No | |
| 44 | 33446 | 54 | F | 0.3 | 0.2 | 150 | Hypo | 60 | 100/70 | I | 64 | 0.9 | 0.7 | 89 | No | No | No | No | No | No | NO | No | |
| 45 | 35322 | 35 | F | 0.23 | 0.12 | 112 | Hypo | 70 | 120/80 | II | 60 | 1 | 0.9 | 112 | No | No | No | No | No | No | NO | No | |
| 46 | 46332 | 45 | F | 40 | 2.1 | >100 | Hypo | 60 | 130/80 | II | 60 | 0.9 | 0.7 | 150 | No | No | No | No | No | No | NO | No | |
| 47 | 47337 | 24 | F | 16 | 0.8 | >150 | Hypo | 68 | 110/80 | II | 65 | 1.2 | 1 | 142 | Yes | Gr II MR + | No | TR + | PR + | PHT + | 40 | Mod | + |
| 48 | 45526 | 26 | M | 34 | 1.53 | 48.7 | Hypo | 60 | 100/70 | I | 62 | 0.7 | 0.5 | 98 | No | No | No | No | No | No | NO | No | |
| 49 | 35746 | 38 | F | 25.6 | 2 | >100 | Hypo | 72 | 120/80 | II | 65 | 0.6 | 0.5 | 98 | No | No | No | No | No | No | NO | No | |
| 50 | 34347 | 40 | F | 150 | 12.8 | 0.32 | Hyper | 112 | 110/70 | II | 60 | 0.8 | 0.7 | 140 | No | No | No | No | No | No | NO | No | |

# Hyperthyroidism Group

| S.No | IP No. | Age | Sex | T3 | T4 | TSH | Status | Pulse | B.P | NYHA | LVEF% | IVS(s) | IVS(d) | LV mass | RWMA | MR | MVP | TR | PR | PHT | TRPG | PE | DCM |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1 | 39786 | 33 | F | 158 | 13.5 | 0.1 | Hyper | 150 | 110/70 | II | 67 | 1 | 0.9 | 112 | No | Tri MR | MVP + | No | No | No | NO | No | |
| 2 | 45672 | 28 | F | 89.3 | 12 | 0.02 | Hyper | 100 | 130/90 | I | 65 | 0.8 | 0.7 | 87 | No | No | No | No | No | No | NO | No | |
| 3 | 39545 | 37 | F | 112 | 14.8 | 0.1 | Hyper | 120 | 120/80 | II | 60 | 0.9 | 0.7 | 114 | No | No | No | No | No | No | NO | No | |
| 4 | 38976 | 22 | M | 200 | 15 | 0.004 | Hyper | 114 | 110/70 | II | 65 | 0.9 | 0.7 | 90 | No | No | No | No | No | No | NO | No | |
| 5 | 44515 | 34 | M | 160.8 | 15.3 | 0.008 | Hyper | 130 | 120/80 | II | 65 | 0.7 | 0.5 | 89 | No | Tri MR | MVP+ | No | No | No | NO | No | |
| 6 | 44345 | 22 | M | 156.8 | 14.3 | 0.05 | Hyper | 120 | 130/90 | II | 60 | 0.7 | 0.5 | 115 | No | No | No | No | No | No | NO | No | |
| 7 | 46543 | 18 | F | 159 | 15.4 | 0.04 | Hyper | 114 | 112/78 | I | 60 | 0.6 | 0.5 | 140 | No | No | No | No | No | No | NO | No | |
| 8 | 42346 | 32 | F | 150 | 11.8 | 0.32 | Hyper | 112 | 120/80 | II | 67 | 0.7 | 0.5 | 90 | No | No | No | No | No | No | NO | No | |
| 9 | 43234 | 29 | F | 162.7 | 12.4 | 0.04 | Hyper | 128 | 120/80 | I | 65 | 0.9 | 0.7 | 87 | No | Tri MR | MVP + | No | No | No | NO | No | |
| 10 | 43562 | 34 | M | 156 | 13.6 | 0.2 | Hyper | 120 | 110/70 | I | 65 | 1 | 0.9 | 92 | No | No | No | No | No | No | NO | No | |
| 11 | 44356 | 35 | F | 160 | 14.8 | 0.28 | Hyper | 130 | 140/90 | II | 60 | 1 | 0.9 | 112 | No | No | No | No | No | No | NO | No | |
| 12 | 44342 | 23 | F | 152.8 | 12.5 | 0.042 | Hyper | 128 | 112/80 | I | 60 | 0.9 | 0.7 | 160 | No | No | No | No | No | No | NO | No | |
| 13 | 43352 | 35 | F | 154.9 | 13.4 | 0.32 | Hyper | 132 | 120/80 | II | 64 | 0.9 | 0.7 | 150 | No | No | No | No | No | No | NO | No | |
| 14 | 45432 | 22 | F | 156 | 14.2 | 0.3 | Hyper | 140 | 112/70 | II | 67 | 0.7 | 0.5 | 98 | No | No | No | No | No | No | NO | No | |
| 15 | 42324 | 32 | F | 154.2 | 13.4 | 0.024 | Hyper | 100 | 110/70 | I | 65 | 0.6 | 0.5 | 95 | No | No | No | No | No | No | NO | No | |
| 16 | 41234 | 30 | F | 162.4 | 15.6 | 0.23 | Hyper | 124 | 130/90 | i | 60 | 0.8 | 0.7 | 78 | No | No | No | No | No | No | NO | No | |
| 17 | 42321 | 35 | F | 158 | 14.3 | 0.34 | Hyper | 132 | 120/80 | I | 65 | 1 | 0.9 | 140 | No | No | No | No | No | No | NO | No | |
| 18 | 34523 | 28 | F | 153.6 | 15 | 0.22 | Hyper | 132 | 110/70 | II | 65 | 0.8 | 0.7 | 90 | No | No | No | No | No | No | NO | No | |
| 19 | 35423 | 32 | F | 165.2 | 16.7 | 0.34 | Hyper | 130 | 120/86 | II | 60 | 0.9 | 0.7 | 87 | No | Gr I MR | MVP+ | No | No | No | NO | No | |
| 20 | 31234 | 28 | F | 154.7 | 15.6 | 0.26 | Hyper | 134 | 120/70 | II | 60 | 1 | 0.9 | 92 | No | No | No | No | No | No | NO | No | |
| 21 | 36543 | 34 | F | 152 | 16 | 0.32 | Hyper | 128 | 130/90 | I | 67 | 0.9 | 0.7 | 112 | No | No | No | No | No | No | NO | No | |
| 22 | 43521 | 32 | M | 156.3 | 15.8 | 0.28 | Hyper | 136 | 112/80 | II | 65 | 0.9 | 0.7 | 112 | No | No | No | No | No | No | NO | No | |
| 23 | 44332 | 32 | M | 154 | 15.8 | 0.33 | Hyper | 140 | 130/90 | II | 65 | 0.7 | 0.5 | 87 | No | No | No | No | No | No | NO | No | |
| 24 | 42334 | 26 | F | 160 | 16.4 | 0.28 | Hyper | 128 | 120/70 | II | 60 | 0.9 | 0.7 | 114 | No | No | No | No | No | No | NO | No | |
| 25 | 45445 | 35 | F | 156 | 15.7 | 0.34 | Hyper | 126 | 110/70 | I | 60 | 0.8 | 0.6 | 90 | No | Tri MR | MVP + | No | No | No | NO | No | |

Hyperthyroidism group contd….

| S.No | IP No. | Age | Sex | T3 | T4 | TSH | Status | Pulse | B.P | NYHA | LVEF% | IVS(s) | IVS(d) | LV mass | RWMA | MR | MVP | TR | PR | PHT | TRPG | PE | DCM |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 26 | 44567 | 35 | F | 150 | 15 | 0.33 | Hyper | 112 | 110/70 | II | 64 | 0.9 | 0.7 | 89 | No | No | No | No | No | No | NO | No | |
| 27 | 45673 | 28 | F | 152.8 | 12.8 | 0.32 | Hyper | 120 | 120/80 | I | 60 | 1 | 0.9 | 115 | No | No | No | No | No | No | NO | No | |
| 28 | 46543 | 22 | F | 154.3 | 13.5 | 0.1 | Hyper | 100 | 130/90 | II | 60 | 1.1 | 0.9 | 140 | No | No | No | No | No | No | NO | No | |
| 29 | 41256 | 36 | F | 162.4 | 12 | 0.004 | Hyper | 120 | 120/86 | II | 65 | 0.9 | 0.7 | 90 | No | No | No | No | No | No | NO | No | |
| 30 | 44563 | 33 | M | 165.7 | 14.8 | 0.006 | Hyper | 114 | 130/70 | II | 62 | 0.7 | 0.5 | 87 | No | Tri MR | MVP + | No | No | No | NO | No | |
| 31 | 45432 | 32 | F | 200 | 14 | 0.05 | Hyper | 112 | 120/80 | II | 65 | 1.1 | 0.9 | 92 | No | No | No | No | No | No | NO | No | |
| 32 | 46554 | 22 | F | 1445 | 15.2 | 0.33 | Hyper | 150 | 140/80 | II | 65 | 1 | 0.9 | 112 | No | No | No | No | No | No | NO | No | |
| 33 | 46654 | 34 | F | 165 | 15 | 0.2 | Hyper | 128 | 130/90 | I | 65 | 0.7 | 0.5 | 160 | No | No | No | No | No | No | NO | No | |
| 34 | 44334 | 22 | F | 150 | 14.2 | 0.05 | Hyper | 130 | 128/74 | II | 60 | 0.7 | 0.5 | 150 | No | No | No | No | No | No | NO | No | |
| 35 | 38765 | 32 | F | 167.8 | 14 | 0.28 | Hyper | 128 | 120/90 | II | 60 | 0.9 | 0.7 | 98 | No | No | No | No | No | No | NO | No | |
| 36 | 33446 | 26 | F | 200 | 14.5 | 0.024 | Hyper | 100 | 120/70 | I | 67 | 0.9 | 0.7 | 95 | No | No | No | No | No | No | NO | No | |
| 37 | 34523 | 32 | M | 150 | 12.8 | 0.34 | Hyper | 124 | 124/78 | I | 65 | 1 | 0.9 | 78 | No | No | No | No | No | No | NO | No | |
| 38 | 33554 | 32 | M | 158 | 14.8 | 0.23 | Hyper | 132 | 130/90 | II | 65 | 1.1 | 0.9 | 291 | No | No | No | No | No | No | NO | No | |
| 39 | 34454 | 53 | F | 90 | 13.4 | 0.22 | Hyper | 130 | 120/90 | II | 60 | 0.9 | 0.7 | 112 | No | No | No | No | No | No | NO | No | |
| 40 | 43225 | 30 | F | 112 | 15 | 0.28 | Hyper | 134 | 130/90 | I | 60 | 0.7 | 0.5 | 150 | No | No | No | No | No | No | NO | No | |
| 41 | 43352 | 40 | F | 200 | 16.7 | 0.33 | Hyper | 136 | 140/90 | I | 65 | 1.1 | 0.9 | 142 | No | No | No | No | No | No | NO | No | |
| 42 | 43354 | 28 | F | 160.8 | 13.6 | 0.28 | Hyper | 124 | 120/80 | II | 60 | 1 | 0.9 | 98 | No | No | No | No | No | No | NO | No | |
| 43 | 45543 | 33 | M | 156 | 13.4 | 0.34 | Hyper | 120 | 124/76 | I | 60 | 0.7 | 0.5 | 98 | No | No | No | No | No | No | NO | No | |
| 44 | 34442 | 32 | F | 162.4 | 14.4 | 0.02 | Hyper | 124 | 120/80 | II | 64 | 0.7 | 0.5 | 140 | No | No | No | No | No | No | NO | No | |
| 45 | 33221 | 34 | F | 160.5 | 16 | 0.1 | Hyper | 112 | 130/90 | II | 67 | 0.9 | 0.7 | 112 | No | No | No | No | No | No | NO | No | |
| 46 | 43322 | 30 | F | 158 | 15.5 | 0.004 | Hyper | 120 | 120/70 | II | 65 | 1.1 | 0.9 | 87 | No | No | No | No | No | No | NO | No | |
| 47 | 45332 | 36 | F | 153.6 | 16.3 | 0.04 | Hyper | 126 | 110/70 | I | 60 | 1 | 0.9 | 114 | No | No | No | No | No | No | NO | No | |
| 48 | 43521 | 28 | F | 154.8 | 14.3 | 0.32 | Hyper | 112 | 120/80 | II | 65 | 1 | 0.9 | 90 | No | No | No | No | No | No | NO | No | |
| 49 | 35544 | 24 | M | 154 | 16.4 | 0.027 | Hyper | 130 | 112/80 | I | 65 | 0.9 | 0.7 | 89 | No | Gr I MR | MVP | No | No | No | NO | No | |
| 50 | 33445 | 35 | M | 148.3 | 15.2 | 0.23 | Hyper | 134 | 120/80 | I | 60 | 0.9 | 0.7 | 112 | No | No | No | No | No | No | NO | No | |